Virginia MEDICAL MONTHLY

OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

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WILLIAM HENRY
WELCH ISSUE

November, 1950

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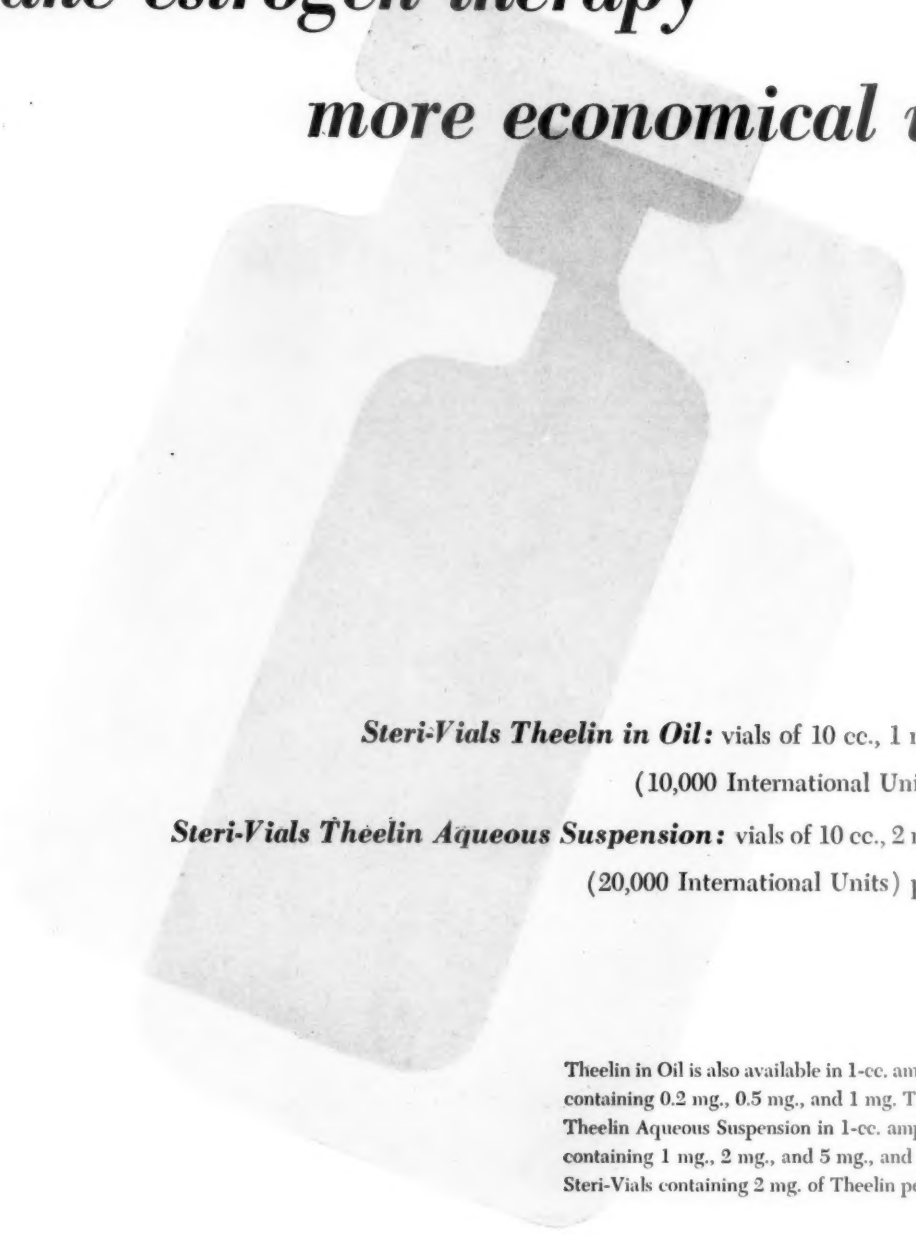

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Virginia Medical Monthly

Vol. 77, No. 11
WHOLE No. 1181

RICHMOND, VA., NOVEMBER, 1950

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25 CENTS A COPY

GUEST EDITORIAL

COOPERATIVE EFFORTS BRING RESULTS

The Virginia Council on Health and Medical Care Links Organizations and Agencies

WE can all be proud of the progress which Virginia has made in the field of health and medical care during the past few years. The term ALL is used because it has been largely through the coordinated efforts, planning, and program of the Virginia Council on Health and Medical Care that much of this progress has been made possible. The Council's membership includes some 43 state-wide and 100 local organizations and agencies representing practically all the voluntary and official groups in the state interested in health. Through them it has awakened the public to its health needs, and has made the public health conscious.

The Medical Society of Virginia has played an important part in the Council's program since its formation in January of 1946. Following the startling facts revealed in the publication of reports by the Virginia Rural Health and Medical Care Survey Committee and the Virginia Advisory Legislative Council, Dr. H. B. Mulholland of the University of Virginia Medical School, then president of the Medical Society, was asked by the Virginia Tuberculosis Association to call together the official and voluntary health agencies in the state for the purpose of coordinating their programs. This was done and the Virginia Council on Health and Medical Care came into existence.

The Council does not attempt to absorb, direct, or control the programs of its highly specialized member organizations, or to establish a competitive health agency. Its purposes are to strengthen through united support a full health program for Virginia, serve as a clearing house on health and medical care problems and programs, assist in eliminating duplication and overlapping, when desirable, and bring statewide and local organizations and individuals together to facilitate joint planning. These purposes are not merely set down in the Council's constitution, but are actively pursued and have become a reality. To quote from a University of Virginia Extension Division Bulletin, "the Council has gone a long way toward proving that, through cooperation, agencies can strengthen a total program without sacrificing their own specialized interests. Indeed many agencies admit that they are stronger as individual units because of the pooling of efforts in areas of common interest."

As part of the Council's educational program, Public Opinion Reports are published when the need arises. These reports give brief, yet clear and concise information pointing out the accomplishments and the unmet needs in the health field. The reports are widely distributed by the member organizations of the Council so that they reach the "grass roots" in every section of the state. Not only is this educational material spread throughout our state, but requests for it come in from all parts of the United States, and even foreign countries. At a recent meeting of the National Health Council the

Virginia Council's published reports were pointed out as being the best example of what good educational material in this field should be like.

There has been a certain amount of interest shown on the part of communities in the state to form local health councils. The State Council has given help and encouragement to these councils when help has been requested. It is their stated policy, however, not to go out to organize councils, but rather to let the initiative for their organization come directly from local need and local enthusiasm.

From time to time the Council undertakes special projects which the membership agrees are worthy of consideration. Recently it set up a committee, under the chairmanship of Dr. W. C. Caudill, to study ways and means of making graduate medical education available to Negroes in AMA approved hospitals of the state, or gaining approval of hospitals where there are at present internships offered to Negroes. Another recent project was the undertaking of the work of the Virginia Health Committee of the Midcentury White House Conference on Children and Youth.

The most extensive and best known project of the Council was started late in 1948 by a committee of physicians, rural sociologists, professional and lay health workers, under the chairmanship of Mr. Henry S. Johnson, Executive Secretary-Treasurer, and Director of Public Relations for the State Medical Society. It was the purpose of this committee to determine the relative need for physicians in rural Virginia, to determine the availability of physicians to settle in Virginia, and to study the possibility of securing information from communities with regard to inducements they might give to have a physician locate in their community. As a continuing function of the committee's work, the Council is now acting as the clearing house for information on communities needing doctors and doctors who have written to the state expressing an interest in establishing their practice in Virginia. As of October 1, 1950, the Council was corresponding with approximately 32 doctors interested in coming to Virginia and 66 communities in the state directly or indirectly requesting doctors. It might be added that all communications from localities requesting doctors are carefully investigated. Detailed information regarding local schools, roads, hospital facilities, housing, etc. which might be of interest to physicians, is kept on file and made available on request. The Council is informing senior medical students at the Medical College of Virginia and the University of Virginia Medical School of the service and information which will be available to them when they complete their internships. It is also contacting the young graduates during their internships here and in other states. It is hoped that with information readily available to them, they will be encouraged to return to Virginia to practice in areas of need, and not be lost to other states.

As part of its unified health program, the Virginia Council on Health and Medical Care tries to make its specialized member organizations see their important place and relationship to the various other programs in the total health picture. After all cancer, tuberculosis, crippled children, heart, polio, etc. all benefit from expanded public health facilities, increased facilities at the medical schools, hospital and health center construction, improved mental hospitals, etc., and vice versa. Health programs are interrelated and cannot be set apart or isolated from one another.

The work of the Virginia Council on Health and Medical Care is becoming increasingly recognized as outstanding in its field. Many other states have written in for its constitution and by-laws, and educational material. Recently Mr. Aubrey D. Gates, Field Director of the Committee on Rural Health of the American Medical Associa-

tion, spent the good part of a day in Richmond getting background material on the Council's program and learning how it functions. In his report to the AMA he wrote, "reiterating what I have already said about the Virginia Council on Health and Medical Care, my appraisal is that it is one of the best, if not the best, State Council in the nation". Mr. Gates pointed out that the Council "is rendering a fine service and especially in the field of promoting better relationships between agencies and organizations".

Fine recognition was paid to the Council in an editorial entitled "Mapping Rural Medicine", which appeared in the *New York Times* on October 1st. It stated, "The Virginia Council on Health and Medical Care offers an example of how to map careers in rural medicine. The Council's surveys have shown where every doctor in the state is located, the extent of his practice, and the need for medical service that is to be met in the community. Its maps are guides to young physicians completing their internships, or to experienced practitioners who would like a breath of country air".

The Council's success can be directly attributed to the fact that it belongs to all Virginians: physicians, farmers, nurses, lawyers, dentists, club women, professional and lay health workers, and others. They are all working together to make it a success, and are bringing better health and medical care to the people of our Commonwealth in this democratic way.

EDGAR J. FISHER, JR., *Director*
Virginia Council on Health and Medical Care

Floral Eponym

Alstonia

ALSTON, CHARLES (1683-1760)

DR. CHARLES ALSTON was educated at Glasgow and Leyden. In the latter University he was under Boerhaave and there he became acquainted with Alexander Monroe. The two Scots returned to Edinburgh and revived the medical lectures at the University. Alston became lecturer in botany and materia medica, and later the superintendent of the botanical gardens. Upon his death he was succeeded by Hope, who edited and published Alston's notes on materia medica. Robert Brown dedicated to him the apocyneous genus Alstonia.

Alstonia is a genus of 30 or 40 species of Asiatic and Australian trees.

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AMERICAN MEDICINE MUST REMAIN FREE*

W. C. CAUDILL, M.D.,
Pearisburg, Virginia.

With this session, we complete a seasonal cycle. In the year that has passed since our last meeting, world events have moved with stunning rapidity. We are just beginning to realize the severity of the sacrifices which the free peoples of the world will be called upon to make during the period ahead.

At this moment, of course, it is difficult to appraise the precise role which will be played by the organized medical profession. Of one thing we are certain, however: If the medical profession of the United States is to render effective service in peace or in war, it must remain free. This is no time to bind the hands of the American doctor with the manacles of socialization.

Whatever the extent or the duration of the present crisis, we cannot afford to jeopardize the continued progress of American medical science. It is now high noon in the 20th century. During the 50 years just passed the medical profession has made astounding advances in all phases of the healing arts. In the field of preventive medicine there has been an enormous diminution in the diseases and deaths of early life, the acute infectious diseases, and maternal and infant mortality. The American community no longer fears an epidemic of typhoid or diphtheria, or smallpox.

In the field of medical research we have witnessed huge strides in the development of specifics. In the fight against bacteria the medical arsenal is being stocked with new and powerful antibiotics. One by one, the scourges of the human body are submitting to the unrelenting efforts of the medical scientist.

In the field of surgery, we have perfected techniques of almost incredible complexity. The clinical experience afforded by two world wars enabled the medical profession to perform near miracles in the healing of wounds and the restoration of diseased and damaged tissue.

The general practice of medicine is on a higher level than ever before. The family doctor has at his disposal diagnostic and therapeutic aids which increase his effectiveness many fold. He has available modern and well-equipped hospital facilities, staffed by trained technicians. Opportunities for medical education were never greater, as our accredited schools turn out increasing numbers of graduates each year. In addition, there are expanding opportunities for doctors to undertake graduate work.

All these things are part of the case history of the past half century. I venture to suggest that in no other branch of human endeavor has there been comparable progress. And I venture to suggest also that the reason for that progress may be found in the fact that during these past fifty years there has been a strong spirit of freedom and individual initiative in the medical profession. Progress in medicine, as in other fields, is best nurtured in a free soil.

What shall we say of the next fifty years? While we do not pretend to assay the future, we do know that we are far from the ultimate. We have only begun to be astounded by medical science.

That future, of course, is conditional. It is ours only if we continue to deserve it. It is ours only if we adhere to the principles of freedom which have guided our steps in the past. We shall lose that future if we permit the heady wine of socialism to dull our sensibilities and destroy our initiative.

I intend this as a solemn warning. The insidious processes of socialism are already threatening not only the medical profession, but all of our free institutions. This year 1950 finds our United States standing virtually alone as the last bastion of constitutional freedom in a world bent on embracing various forms of collectivist society. But our own anchors are no longer firm. We, too, are drifting, faster than most of us realize, toward the yawning vortex of the welfare state.

The great labor leader, Samuel Gompers, warned in an address given more than thirty years ago: "I bid you have a care in all these attempts to regulate the personal relations and the normal activities of the citizenship of our country before it is too late. For a mess of pottage, under the pretense of compulsory social insurance, let us not voluntarily surrender the fundamental principles of liberty and

*Address by the President before the annual meeting of the Medical Society of Virginia at its meeting in Roanoke, October 9, 1950.

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freedom."

We must understand that the body politic, like the human body, can be ruined by overmedication. In a class conducted by Sir William Osler, the students were asked how much of a certain drug should be given to a patient. One student promptly answered, "Four grains", and none of the others dissented. Sir William continued discussing the case symptoms, and a few minutes later he was interrupted by the same student, who asked permission to change his answer. Sir William gravely consulted his watch and said, "I'm sorry, young man, but your patient has already been dead for three minutes."

We see much the same thing happening today in our nation's economic life. Fuzzy-minded politicians and addle-brained theorists have the idea that because one grain of socialization has proved helpful, four grains well bring us utopia. Because it is necessary for the government to undertake a few essential services, they feel that the government should perform more and more services and ultimately attend to all our human wants and needs.

The advocates of government by overdosage are busy with their propaganda. They have introduced numerous schemes to set up a system of compulsory health insurance. They insist that America follow the lead of nations whose health standards are far below our own. We hear the assertion made that medical care is too expensive for the average American family. The prescription for this situation is to establish a vast bureaucratic monster, to administer a compulsory health program at a cost estimated variously between ten and twenty billion dollars a year.

The whole thing is a patent nonsense. Why does America need socialized medicine when she leads the world in medical research and scientific development? Why does America need socialized medicine when we have more doctors, dentists, nurses, and hospitals per capita than any other country? Why does America need socialized medicine when American citizens enjoy a long and continually lengthening life span? Why does America need socialized medicine when there are enrolled today in voluntary medical insurance programs more than seventy million people, in addition to the tens of thousands who participate in industrial, fraternal, and labor-health programs and private group-clinics?

Why indeed? The whole idea is a dismal distortion of logic. It is blandly declared that 325,000 people die needlessly each year, and the implication is that if their medical care were only turned over to the government, the figure would be mysteriously reduced to zero. But when we examine the figures, we find that some forty thousand of these deaths are deaths by accident—and no health insurance program I know of has ever been effective against accidental death. Another one hundred thousand of these deaths are caused by cancer and heart disease—diseases which are the inevitable accompaniment of old age. Communicable diseases account for one hundred and twenty thousand of the deaths. We know that these can be prevented because we are preventing them in large measure. Through the efforts of the medical profession over the past fifty years illnesses and fatalities from these diseases have been reduced at an astounding rate.

The arguments advanced by the proponents of socialized medicine are so feeble that no informed person can be convinced by them. But there is one argument that the socializers have not yet mentioned. If they wanted to be fair about it, they would call as their number one witness a man named Nikolai Lenin, the father of the police state. "Socialized medicine", said Comrade Lenin, "is the keystone of the arch of the socialistic state."

Here is the crux to the whole issue. Socialized medicine is the entering wedge. It is the decoy. It would be the means of creating a gigantic new army of bureaucrats, replete with regional administrators, inspectors, coordinators, and collectors. It would elevate political hacks to serve as local czars in every city, town, and village in the country. The healing art would degenerate into a political pill mill, with every doctor, dentist, nurse, and technician reduced to the status of a lackey on the public pay roll.

The inevitable accompaniment of socialized medicine would be dictatorial control over the life of the individual. Members of the medical and dental professions, of course, would be the first to feel the long arm of government control. They would be placed in the same position as the doctors and dentists in Great Britain, whose Labor Government has instituted an all-inclusive Compulsory Health Insurance Program. Impartial observers agree that British doctors are so desperately over-worked and so burdened with bureaucratic red tape that they are losing all incentive for adequate practice. "Quetopia" is the term that has been applied to the British medical

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phenomenon. The "something for nothing" philosophy has grasped the British people to such an extent that doctors are forced to waste valuable time dealing with patients whose ills are either imaginary or negligible, while truly deserving patients await their turn.

Yet there are those who would have us destroy the soul of American medicine by following the British example. There are those who would have us adopt an expensive, cumbersome, inefficient, and dictatorial scheme which would jeopardize the high medical standards of the present and the progress which we may expect in the future. There are those who would play fast and loose with the fiscal structure of the United States by instituting a tax levy of from three to ten per cent on every paycheck up to $4,800 a year, at a time when our national economy demands rigid control over expenditures in order to fulfill our military and diplomatic obligations. There are those who would destroy the sacred personal relationship between doctor and patient and inject the government as an unwelcome, prying third party.

You and I know who are the chief advocates of political medicine. They are the bureaucrats like Oscar Ewing, of the Federal Security Agency, who see in socialized medicine a means of expanding their power and insuring lush political plums for themselves and their political satellites. They are the powerful and arrogant labor leaders who view socialized medicine as an additional means of control over the vast army of American workers. They are the Communists and their dupes who view socialized medicine as the "keystone of the arch of the socialistic state."

We may be thankful that the vast majority of the American people have not fallen prey to the fuzzy thinking of these elements. The front of opposition to socialized medicine is being rapidly expanded to include such influential organizations as the American Legion, the National Grange, the Veterans of Foreign Wars, the General Federation of Women's Clubs, the American Bar Association, the U. S. Chamber of Commerce, the American Council of Christian Churches, the American Farm Bureau Federation, and numberless other groups and individuals who have the courage to stand up for fundamental Americanism and refuse to be lured by the siren song of socialization.

The stand taken by these organizations gives the lie to any assertion that the American Medical Association constitutes the sole opposition to socialized medicine. The American Medical Association is only one of many organizations which have banded together in this crusade to maintain freedom in American medical practice.

But let it not be assumed that the medical profession is content with a negative approach to the economic aspects of medical practice. The American Medical Association is a pioneer in the development of voluntary prepayment plans. The approach of the A.M.A. to the nation's medical problems is on the broadest possible scale, embracing mental and physical health, disease prevention, medical treatment as needed, prolongation of life, and research for the advancement of medical science. It includes also a program of nutrition, recreation, prenatal care, child welfare, construction of hospitals and diagnostic centers, and health education. Prepaid medical service plans already in operation under the sponsorship of organized medical societies have a membership of seventy million and it is estimated that the number will be increased to ninety million within the next three years.

This is ample evidence that America's problems of medical economics are well on the way toward being solved—solved not through any intervention of the politicians, but solved through the efforts and endeavors of the medical profession itself, with the cooperation of various lay organizations, acting under the time-tested principles of American free enterprise.

Ministers to the sick have a reputation which neither time nor calumny can tarnish. Even the caustic skeptic Voltaire could find it in his heart to write, "Nothing is more estimable than a physician who, having studied nature from his youth, knows the properties of the human body, the diseases which assail it, the remedies which will benefit, exercises his art with caution, and pays equal attention to the rich and poor." Similarly, Robert Louis Stevenson wrote that the physician has an opportunity as no other class of men to be the flower of our civilization.

This is an opportunity we do not hold lightly. So long as the doctor remains free, so long as the society he serves enjoys a maximum of liberty, we have the promise that American standards of health will continue to be the highest in the history of the world.

SYMPOSIUM ON HEART DISEASE†

MANAGEMENT OF THE COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION*

RENO R. PORTER, M.D.,
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and
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There are numerous complications that may occur during the course of acute coronary occlusion with myocardial infarction, all of which tend to make a serious situation all the more precarious. These complications may involve the heart itself or may occur in other organs. This paper deals with those complications involving the heart or originate within the heart. The more important of these complications specific to the heart are shown in the table. These data are taken from 222 cases of acute myocardial infarction seen at the Medical College of Virginia Hospitals from 1938 to 1947, inclusive, and at the McGuire Veterans Administration Hospital from April, 1946, to September, 1949. The overall mortality for this group of patients was 24%. In this table are listed, in the order of frequency, the more common complications occurring during the course of acute myocardial infarction. Since we are concerned at this time with episodes that occur during the acute stage, only the events during the first three to four weeks are noted. The chronic residuals or late events of myocardial infarction are not within the scope of this paper.

ACUTE MYOCARDIAL INFARCTION

	Number	*Mortality*
Total Series	222	24%
With Important Complications:		
Congestive Heart Failure	33	42%
Serious Arrhythmias	29	42%
Shock	27	74%
Embolism	10	80%
Total	99	55%
Without These Complications	123	11%

Table I: Important complications occurring during the course of acute myocardial infarction.

It is seen that congestive failure occurred in 33 cases, 14 of whom died, giving a mortality of 42%. Serious arrhythmias, which include auricular flutter, auricular fibrillation, supraventricular tachycardia, ventricular tachycardia, and ventricular fibrillation, occurred in 29 instances, 12 of whom died, giving a mortality of 42%. Shock (systolic pressure below 90) occurred in 27 cases, 20 of whom died, giving a mortality of 74%. Embolic phenomena occurred in 10 cases, 8 of whom died, with a mortality of 80%. There are other cardiac complications that may occur, but these are the most important from the point of view of incidence and seriousness. It is seen that the mortality from myocardial infarction is definitely greater when these disturbances occur. This is illustrated in another manner by the 123 cases in this series which did not have any of the above-mentioned complications. Only 13 of such cases died, giving a mortality of 11%. Many of this group died suddenly, possibly from some of the above complications, but we had no definite evidence to this effect and so they are placed in the group without complications. It is probable that this mortality figure would be even lower if all the facts were known.

These data do not allow strict statistical analysis because of many factors, the more obvious of which is that one patient may have had more than one of these complications. In addition, we cannot be sure that the particular complication noted was the actual cause of death. Nevertheless, they indicate in a general way that their occurrence definitely increases the hazards of myocardial infarction.

†Three papers presented at the 1949 session of the Medical Society of Virginia and published together upon request as a Symposium.

*Read before the annual meeting of the Medical Society of Virginia, at Fort Monroe, October 11, 1949.

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This work supported in part by the Thalhimer Research Fund.

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Since we are unsuccessful, so far, in preventing myocardial infarction in most instances, the least we can do is attempt to minimize its hazards once it has occurred. This involves many therapeutic approaches, but it seems to us that especial efforts toward the prevention and treatment of the more significant complications should be particularly fruitful. It is the purpose of this paper to discuss certain aspects of the management of these complications. It is possible to prevent some of these complications and this, of course, is the therapeutic ideal. Others, however, occur in spite of prophylaxis or have no particular prophylactic treatment. When this occurs, we must use all therapeutic measures at our disposal to bring to an end this unfavorable event or minimize its adverse effects on the heart and circulation.

Congestive Heart Failure. Congestive heart failure developing during the course of myocardial infarction is the most frequent complication. It is more likely to occur if hypertensive heart disease is associated with the coronary disease. There is nothing particular that can be done to prevent its occurrence. The patients are already at bed rest and oxygen is being given, at least for the first few days. These measures are routine in acute infarction and certainly tend to prevent failure by conserving and nourishing the heart. In general, its management is about the same as when failure occurs under other circumstances. The patients are already at maximum rest. Oxygen by tent, mask, or nasal catheter should be administered. A low salt diet should be used since this has a definite place in reducing fluid accumulations. Formerly digitalis was considered by some to be contraindicated in acute myocardial infarction. Digitalis does have an irritative and stimulant action which might tend to produce ectopic rhythms and rupture of the myocardium from a more vigorous contraction. Certainly this drug should not be given unless congestive failure is present, but under such circumstances it is definitely indicated. The possibility of more vigorous contractions to the point of myocardial rupture has been entirely discounted and actually presents no hazard. The possibility of initiating ectopic rhythms is very real since it has been shown that a decrease in coronary blood flow increases the susceptibility of the myocardium to the irritable effects of digitalis. For this reason it is advisable to give quinidine, grains 3-6, every two to four hours while digitalis is being given. There is a further indication for quinidine being given along with digitalis. Digitalis given alone when auricular fibrillation and congestive failure occur in myocardial infarction has resulted in the past in a high percentage of fatal emboli. Of course, with the current use of anti-coagulants, this hazard probably is greatly reduced. However, the first consideration is sufficient to urge the use of quinidine whenever digitalis is being given. These measures, low salt diet and digitalis with quinidine, usually suffice to restore compensation. Occasionally, diuretics, either zanthine or mercurial, may be needed. In the use of this latter, caution must be exercised not to use mercurials too extensively with a low salt diet, especially when there is associated renal disease. This may result in severe salt depletion and its consequent hypotension.

Serious Arrhythmias. In regard to the more serious arrhythmias, certain general statements can be made. Myocardial infarction renders a heart more irritable and more sensitive to the action of stimulating agents, such as tobacco, digitalis and others. Such increased irritability and sensitiveness makes the heart more prone to develop ectopic rhythms, which are detrimental in themselves. They cause over work for the heart because the beats may be both rapid and ineffectual, with the heart expending excess energy to no benefit. Further, there may be such ineffectual beating of the heart that there is functional cardiac standstill and the patient dies, as in ventricular fibrillation. Therefore, it is important to prevent such arrhythmias or control them properly once they occur. The prevention of these arrhythmias can be attained in most instances by the use of quinidine sulfate in the dose of 3-6 grains three times daily, or perhaps as often as every two to three hours. There are some who advocate the institution of such prophylactic therapy in every case of acute myocardial infarction. We think this is hardly justified, and institute prophylactic quinidine only when premature beats, whether supraventricular or ventricular, occur. These extrasystoles are not particularly harmful in themselves, at least their occurrence does not increase the mortality, but they are in many instances the forerunners of a more serious ectopic rhythm. We likewise advise the use of prophylactic quinidine, when digitalis has to be used in a case of acute myocardial infarction. As mentioned above, infarction increases the sensitivity of the heart to

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the stimulating effects of digitalis and ectopic rhythms are more likely to arise under these circumstances. Quinidine depresses this irritability and tends to prevent such disorders. Finally, smoking should be prohibited, not only because of its possible effect on the coronary circulation, but to eliminate a possible stimulant to the development of an ectopic rhythm.

Should ectopic rhythms develop, specific measures are needed. The more commonly occurring disorders are auricular flutter, auricular fibrillation and ventricular tachycardia. Auricular fibrillation is in many instances a transient phenomena during the course of myocardial infarction. For this reason, it is best to wait twenty-four hours before the institution of anything more than prophylactic quinidine. If it should persist more than twenty-four hours, it is more likely to continue and attempts should be made to abolish it if the heart is compensated. This can be accomplished in most instances with quinidine, grains 6, every two hours for five to seven doses. Careful check of the heart should be made before each dose to see whether normal rhythm has been restored or adverse effects are resulting. If the rhythm is not restored to normal after such a course, or if failure develops, further attempts to break the rhythm with quinidine should be abandoned and the patient digitalized.

Auricular flutter is a much more difficult rhythm since it tends to be persistent and is usually unresponsive to quinidine. Fortunately, this does not occur so often as auricular fibrillation. It is probably better to go ahead and digitalize the patient at the onset. Following digitalization, the rhythm may revert to normal, probably a spontaneous phenomenon. At other times, the flutter is converted into fibrillation, which may revert to normal rhythm upon discontinuing the digitalis. In most instances, however, digitalis has no particular effect on the flutter itself, but does increase the block between the auricles and ventricles. This results in slowing the ventricular rate to about normal and relieves the stress of a rapid heart action on the heart.

Ventricular tachycardia is a very serious arrhythmia since it can lead to death rather rapidly if something is not done about it promptly. It is made more difficult by the fact that digitalis, in many instances, produces this ectopic rhythm and hence is contraindicated in treatment. So we are left with only quinidine as a therapeutic agent. This can be given as described above, except that we will risk giving 6 grain doses over a longer period of time since the situation is critical. However, unless results are obtained, this cannot be continued for too long by mouth because the patient's condition usually becomes near fatal in 18 to 24 hours. Under such circumstances, more heroic measures are needed which consist of giving quinidine intravenously. This is a very dangerous procedure, but is warranted by the critical state of the patient. He will die shortly if normal rhythm is not restored. Intravenous quinidine can be prepared with some difficulty by dissolving 15 grains in about 300 cc. of distilled water. A special preparation for intravenous use, such as Quinidine Lactate (Lilly) can be used. This comes in ampules containing 0.65 gm. of quinidine lactate which is diluted to 50 cc. with 5% glucose. These preparations are given by slow intravenous drip with the physician's finger constantly on the patient's pulse. At the first break in the rapid pulse rate, the drug is stopped. Failure to stop the drug at the instant of effect may result in death of the patient from quinidine poisoning.

Shock. The development of shock is a fairly common occurrence and is the mechanism of death in many cases of myocardial infarction. This occurs, ordinarily, within the first twelve to twenty-four hours of the onset of the infarction. The patient's blood pressure drops, his skin becomes cold and clammy, the heart sounds are faint, the pulse is weak and thready. The course is fairly rapidly downhill with falling blood pressure, oliguria or anuria, unconsciousness and death in a day or so. Formerly, we stood anxiously by and watched the inevitable occur. We recognized the shock state, but were afraid to use the recognized treatments for shock, other than keeping the patient warm. Stimulants in the form of adrenalin were sometimes advised, but now it is believed that they do more harm than good. The use of intravenous blood or plasma was avoided for fear of overloading an injured heart and precipitating further complications such as congestive failure. However, it has come to be realized that the shock state, while predominantly central in origin, nevertheless initiates a vicious cycle which is injurious to all organs in the body, but is particularly bad for the heart. The fall in blood pressure lowers the head of pressure at the coronary orifices which in turn

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diminishes the blood flow through these arteries. This is detrimental not only by diminishing the flow of blood through the arteries that are open, but also by impairing the development of a collateral circulation around the occluded artery. All of these factors lead to greater myocardial ischemia and probably greater areas of infarction. This in turn produces further shock and the cycle is repeated. Therefore, vigorous treatment is as imperative in treating the shock of myocardial infarction as that due to other causes. Blood and plasma transfusions have been found to be distinctly beneficial under these circumstances and now we have no hesitancy in instituting this therapy when shock occurs in myocardial infarction. About the only precautions taken are to run the transfusion rather slowly and to watch the patient a little more closely than we do under other circumstances. It is very gratifying to observe the patient come out of his critical state with a rise in blood pressure, a brightening of consciousness and improved renal function. It is a source of additional encouragement to realize that the coronary circulation is also much improved by this treatment. We have noted no ill effects from such a procedure. This gratifying response has caused us to consider the possibilities of arterial transfusions under these circumstances. This type of procedure can cause immediate increase in aortic pressure and with much less blood than is required by the venous route. In addition, this technique would obviate the potential danger of overloading the damaged heart before the beneficial effects of increased arterial pressure are attained. Arterial transfusions, however, present certain technical difficulties at the present time. Nevertheless, it is theoretically very promising and studies are currently underway to eliminate the technical difficulties and to evaluate its efficacy.

Embolic Phenomena. Embolic phenomena in the past have always been a serious threat during the course of acute myocardial infarction. Its incidence was not too frequent, but in nearly all instances it was fatal when it occurred. Preliminary results so far seem to indicate that the use of anticoagulants have reduced the possibility of this serious complication and should be used routinely in order to prevent embolic phenomena. A more detailed discussion of this therapy is to be given in the paper that follows. In this connection, I might say that we have seen no instances of embolism occurring in patients with adequate anticoagulant therapy. The ten cases noted as having embolism occurred in patients with no anticoagulants or inadequate amounts. As has been mentioned, the use of quinidine along with digitalis when treating auricular fibrillation in myocardial infarction has been shown to reduce the incidence of emboli. So both of these measures should be used as prophylaxis to prevent this serious complication. If embolism occurs, the embolus should be removed surgically as quickly as possible if it is in an accessible artery. Sympathetic block should also be used if the embolus lodges in an extremity artery. Papavarine in doses of grains 1½ should be given every four hours whether or not operation can be performed. On the whole, this is a very distressing condition, and treatment in many instances is unsatisfactory. It is much better to try to prevent them by the measures mentioned above.

CONCLUSION

Acute myocardial infarction is a serious catastrophe in the life of any person. It may be an immediate threat to life as well as imply an unfavorable outlook for the future. However, the chances of surviving the acute episode are very good provided the complications of serious arrhythmias, congestive failure, shock and embolism do not occur. As a corollary, the occurrence of these complications greatly decreases the chances of survival. We should, therefore, be on the alert to prevent these complications and to treat them adequately should they occur. In this way, we can hope to reduce the immediate hazard of this condition.

DISCUSSION

DR. JULIAN R. BECKWITH, Clifton Forge: Dr. Porter has covered the subject so thoroughly that there is very little left to be added.

The management of myocardial infarction consists to a great extent of the prevention or treatment of the complications of the disorders. To date we know of no way to prevent coronary sclerosis, which leads to coronary thrombosis with resulting myocardial infarction. Perhaps we can prevent some instances of myocardial infarction by cautioning people who probably have coronary sclerosis to avoid undue exertion which would result in relative coronary insufficiency followed by myocardial necrosis. Cautioning people about keeping their weight down would also probably reduce the incidence of coronary disease. However, the management of the disease lies in the management of its complications.

Dr. Porter has shown that heart failure carries a high mortality, and he has pointed out that this should be

controlled. We have used mercurial diuretics and a low salt diet on several occasions without digitalis; however, we do not hesitate to use digitalis in the heart failure that follows myocardial infarction if necessary.

Ventricular tachycardia was mentioned as a serious complication, and it might be of interest to mention a patient that we had recently who has this disorder for nine days before the rhythm reverted to normal. Quinidine was given in doses of 9 grains every 3 hours and the ventricular rate decreased from 150 to 100. Concurrently the blood pressure and peripheral circulation became normal, and urinary output increased. Finally quinidine was stopped and the rhythm returned to normal. This may have been coincidental.

It appears that one of the most important advances that have been made recently in the management of myocardial infarction is the use of blood and plasma to control shock. The effectiveness of this has been beautifully shown in the laboratory by Dr. Prinzmetal, whose experiments demonstrated that a ballooning out of the infarcted area occurred in shock and that this could be corrected by the administration of blood in sufficient quantities to control shock. We have had no clinical experience with this as yet but intend to employ it at the next opportunity.

Finally, I should like to emphasize, as Dr. Porter did, that most patients do well with myocardial infarction and that careful attention to details of the management of complications will save many lives.

This paper is an excellent summary of a timely subject, and I appreciate the opportunity of discussing it.

CHAIRMAN OUTLAND: It occurred to me (though of course I shall not attempt to dictate it) that since the subjects of these papers are so closely related we might have the primary discussion on each one immediately after its presentation and then have the general discussion after all of them are read. May I have a suggestion on that?

A MEMBER: I move that we do that, Mr. Chairman.

CHAIRMAN OUTLAND: Is there any objection to that? If there is, don't hesitate to voice it. Since I hear none, we will proceed on that plan.

THE USE OF ANTICOAGULANTS IN THE MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION—
A Report of Sixty Cases*

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and
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In 1938, Solandt and Best[1] described the use of heparin experimentally in dogs, as a preventive for thrombosis of the coronary artery but its clinical implications were not followed up because of the expense, the limited supply at the time, and the impractibility of administration. This drug may be given intravenously as the sodium salt or intramuscularly as Pitkin's menstruum. When given intravenously frequent determinations of the coagulation time of the blood must be carried out; thus, it is hardly practical for routine use. Intramuscular heparin has been used in coronary thrombosis and the results are said to be encouraging.[2] However, the drug is expensive and dicumarol appears to be the drug of choice at the present time.

Dicumarol was found by Professor Karl Link, *et al.*, to be the active principle of spoiled sweet clover which causes hemorrhagic disease in cattle.[3] This material which was synthesized by them is a white crystalline solid, soluble in alkaline solution but only slightly soluble in water. It depresses the prothrombin concentration of the plasma and in this way prevents clotting from taking place.

CHART I

PROTHROMBIN + THROMBOPLASTIN
+ CALCIUM = THROMBIN
THROMBIN + FIBRINOGEN = FIBRIN

Chart I shows briefly the well known clotting scheme. Prothrombin is formed chiefly in the liver and circulates in the blood. This combines with thromboplastin, which is found in many tissues, in the presence of calcium to form thrombin, which in turn combines with fibrinogen to form fibrin.

Dicumarol has a delayed action so that a given dose will usually not become effective for 48 hours and its effect can be nullified in a few hours by large

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Read before the annual meeting of the Medical Society of Virginia at Fort Monroe, October 9-12, 1949.

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doses of synthetic vitamin K intravenously or by the administration of fresh whole blood.

In 1942, Allen, *et al.*,[4] and Wright, *et al.*,[5] described the use of dicumarol in the management of thromboembolic disease, particularly in the prevention of extension of the thrombosis in the veins of the extremities and thereby preventing pulmonary embolism. This drug was found to be safe, cheap, easy to administer and effective when given by mouth.

In 1946, reports of the use of dicumarol in the management of myocardial infarction began to appear in the literature[6] and it seemed likely that the mortality of this disease could be considerably reduced by the routine administration of this drug. It has been well known for some time that a large part of the mortality of myocardial infarction was due to the development of various thromboembolic phenomena and Table I shows a tabulation of several series of cases of myocardial infarction with the incidence of thromboembolic complications. The incidence of thromboembolic complications is seen to vary in the different series but it is apparently substantial. Of 2,127 cases, 325 or approximately 15% of the total number of cases had thromboembolism. Since the mortality from this alone is considerable, measures that would reduce the incidence of these complications would obviously reduce the mortality of myocardial infarction.

Table II shows the reduction in mortality in the series in which dicumarol was used. This reduction in mortality appears to be chiefly due to the decrease in the thromboembolic phenomena. In Wright's series of 800 cases,[12] death not preceded by a thromboembolic incident occurred in 14% of the control as compared with 12% of the treated group; whereas, the total mortality was 24% and 14.9% respectively. This report also demonstrated that the drug was useful in each of the first four weeks and appeared to prevent extensions of the original infarct, as well as venous thrombosis, cerebral emboli, and peripheral emboli. It is most effective in patients over 50 years of age.

METHOD OF ADMINISTRATION

Dicumarol is given by mouth in doses that are determined by its effect on the prothrombin time of the plasma. This determination must be done daily and the prothrombin activity must be kept within the therapeutic range in order for the drug to be effective. If the prothrombin level drops too low, serious hemorrhagic complications may develop. It is, therefore, imperative that daily prothrombin levels be known.

TABLE I
THROMBOEMBOLIC PHENOMENA IN MYOCARDIAL INFARCTION

Source	No. of Cases	No. With Thromboembolic Phenomena	%	Outcome
Conner and Holt[7]	287	42	14.6	
Master, Dack, and Jaffe[8]	500	29	5.8	6% were fatal
Nay and Barnes[9]	100	18	18	
Peters, Guyther, and Brambel[6c]	60	10	16.6	16% were fatal
Hellerstein and Martin[10]	160	73	45.0	45 contributed to death
Mintz and Katz[11]	572	52	9.9	55.8% died
Wright, *et al.*[12]	368	83	25.0	10% died
Present Series	80	8	10.0	75% died
Total	2,127	325	15.2	

The method of Quick[14] is satisfactory for determination of the prothrombin time. This consists essentially of adding rabbit's brain as a source of thromboplastin, and calcium chloride to the patient's plasma. The clotting time of this plasma is observed and the prothrombin activity is reported in seconds. A normal control determination is done as a comparison with each test. The average normal prothrombin time is 12 seconds. A therapeutic level is considered to be between thirty and sixty seconds.

In administering dicumarol, a therapeutic level is obtained as quickly as possible though there is usually a delay of about 48 hours after the drug is given before this level is reached. The initial dose is 300 mg., and if the prothrombin time is below 20 seconds the following day, 200 mg. are given. The prothrombin time is then usually between 20-30

TABLE II

MORTALITY OF MYOCARDIAL INFARCTION

Source	Control No. Cases	Control No. Died	Control %	Dicumarol Treated No. Cases	Dicumarol Treated No. Died	Dicumarol Treated %
Nichol and Page[6b]	--	--	---	44	8	18
Peters, *et al.*[6c]	60	13	21.6	50	2	4
Parker and Barker[6d]	---	--	---	50	5	10
Mintz-Katz[11]	572	125	21.8	--	--	--
Greisman and Maurer[13]	100	35	35	75	7	9
Wright, *et al.*[12]	368	87	24	432	64	15
Present Series	80	21	26.5	60	9	15
Total	1,180	281	23.8	711	95	13.3

seconds and 100 mg. are given. However, the size of all doses after the initial dose depends on the prothrombin activity. The response of individuals may vary from day to day and different individuals react differently. Some are hyperreactors; that is, they will have a marked reduction in prothrombin activity following the initial dose and others are hyporeactors; that is, they will require larger amounts of the drug before an effective level is reached.

In determining the dosage of dicumarol for a certain day, the previous reaction to the drug as well as the previous day's levels are considered. If, for example, the prothrombin time was 35 seconds, one would hesitate to give any more if the level had been increasing during the preceding 2-3 days, while if it had been decreasing and was on a downward trend, 50-75 mg. would be given.

Dicumarol is given to patients for four weeks from the time the infarction occurred or from the time of the last thromboembolic episode. The drug is then stopped and the patient is allowed to gradually increase his activities. Even though the patient is first seen two weeks after the infarction has occurred, the drug is administered because it has been shown[12] that the incidence of thromboembolic complication is still high during this period.

CONTRAINDICATIONS

The most important contraindication is the inability to do daily prothrombin determinations while the drug is being administered. The other contraindications are:

1. Liver damage which might result *per se* in decrease in prothrombin activity.
2. Blood dyscrasia with bleeding tendencies.
3. Ulcerative lesions of the gastro-intestinal tract.
4. Renal insufficiency.
5. Vitamin K deficiency.

COMPLICATIONS

The only known complication is hemorrhage, which can be serious. Fortunately this did not occur in our series. The treatment of hemorrhage centers around the administration of synthetic vitamin K and transfusions of whole blood. Seventy-two mg. of vitamin K given intravenously will usually cause a lowering of the prothrombin time to normal within a few hours. Fresh whole blood is also useful in controlling hypoprothrombinemia, the amount depending on its effect on reduction of the prothrombin time.

PURPOSE OF THE PRESENT STUDY

Our clinical impression after using dicumarol for

TABLE III

CONTROL GROUP VS. DICUMAROL GROUP
AGE GROUPS, MORTALITY

Control Group Age Groups	No.	Died	Mortality	Dicumarol Group Age Groups	No.	Died	Mortality
30-39	3	1	33.3%	30-39	1	0	0
40-49	12	1	8.3%	40-49	8	0	0
50-59	26	4	15.4%	50-59	14	0	0
60-69	26	9	34.6%	60-69	25	5	20%
70-79	12	5	41.6%	70-79	11	3	27.2%
80-89	1	1	100%	80-89	1	1	100%
Total	80	21	26.25%	Total	60	9	15%

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about eighteen months was that the mortality was considerably reduced; hence, it was decided to compare the mortality of those treated patients with a group that had been previously reviewed,[15] who had not been given dicumarol.

A total of 140 patients are included. Eighty make up the control group and sixty the treated group. The former were admitted to the hospital from July, 1941, through September 1, 1943, and from January 1, 1946, through September 1, 1947, so were not consecutive cases. The treated group consisted of sixty patients who were admitted from January 1, 1948, through July, 1949. Both groups contained white and negro patients. The diagnosis of myocardial infarction was made by clinical observation as well as by characteristic electrocardiographic changes. They were all studied and managed according to a plan previously outlined,[15] in addition to which the treated group was given dicumarol.

RESULTS

As can be seen in Table III the distribution according to age groups is about the same in each series. There are not enough cases under forty years of age to make a statistical comparison, but it is striking that no deaths occurred in the treated group. The comparison made in the groups between 50-59, 60-69, 70-79 shows that the mortality is significantly reduced in the treated series.

THROMBOEMBOLISM AND EXTENSION OF MYOCARDIAL INFARCTION

Table IV depicts the thromboembolic complications that occurred in the two groups of patients. A total of 9 complications occurred in 8 patients in the control group. Six had pulmonary infarctions of whom four died. One had cerebral embolism, one had peripheral embolism, and one had peripheral thrombosis all of whom died. The patient who had peripheral venous thrombosis died with pulmonary embolism.

Thus, of the eight patients, six died, a mortality of 75%.

TABLE IV
THROMBOEMBOLISM AND EXTENSION OF MYOCARDIAL INFARCTION

Control Group				Dicumarol Group			
Age Groups	No.	Died		Age Groups	No.	Died	
30-39	2	1	Pulmonary Infarct, L.* Per. Emb., D.*	30-39	0	0	
40-49	1	1	Pulmonary Infarct D*	40-49	0	0	
50-59	1	1	Perip. Thrombosis and Pulmonary Infarct D*	50-59	0	0	
60-69	2	1	Cerebral Embolism, D.* Pulmonary Infarct, L.*	60-69	1	0	Extension of Infarction
70-79	2	2	Pulmonary Infarct D* Both died.	70-79	0	0	
80-89	0	0		80-89	0	0	
Total	8	6		Total	1	0	

Control Group	No.	Result
Pulmonary Infarction	6	4 died
Cerebral Embolism	1	1 died
Peripheral Embolus	1	1 died
Peripheral Thrombosis	1	1 died

*L.—Lived
*D.—Died

In the treated group, one had an extension of the infarction but no other thromboembolic complications occurred. This patient was given dicumarol in the usual manner after the initial infarction and on the 13th day he developed pain again and the electrocardiogram showed changes indicative of an extension of the infarct. Leukocytosis developed and the sedimentation rate subsequently became elevated again. On reviewing his record, it was found that the prothrombin time was not within the therapeutic range on the three days previous to the extension. After this second episode of infarction in this patient, dicumarol was given for one month. The prothrombin level was kept within the therapeutic range and he had no further trouble.

TABLE V
RESULTS OF TREATMENT WITH DICUMAROL IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Sources	No. of Patients	Deaths	Percent Deaths
Parker and Barker[6d]	50	5	10
Peters, Guyther, and Brambel[6c]	50	2	8
Wright, *et al.*[6a]	432	64	15
Greisman and Maurer[13]	75	7	9
Nichol and Page[6b]	44	8	18
Present Series	60	9	15
Total	711	95	13.3

TOTAL MORTALITY

Nine deaths occurred in our series of 60 cases treated with dicumarol. This mortality of 15% compares favorably with those of other series and with the average mortality of the combined group. Table V.

ANALYSIS OF DEATHS

Table VI shows that all but two of the patients who died had satisfactory prothrombin levels and none developed signs of thromboembolic phenomena. Six died with congestive heart failure, two continued to have severe pain and died during the first week after the infarction occurred. One died suddenly on the 13th day. Autopsy revealed an infarct of the subendocardial area of the myocardium of the left ventricle, about two weeks old. There was no evidence of cardiac rupture or of thromboembolic complications. Autopsies were done on three other patients. Each had congestive failure, but in none were thromboembolic complications found. This point is important in that it is well known that a large number of patients who die with congestive failure have thromboembolic disease.

One patient died six weeks after he had the infarction. He had been difficult to manage and got out of bed a number of times. Auricular fibrillation was present on admission. On the third day, a loud systolic murmur was heard, congestive failure followed, and digitalization was carried out. Pneumonia also occurred as a complication which was controlled by penicillin. Death finally occurred in congestive failure. Autopsy showed a perforation of the interventricular septum.

Three of the patients who died had previous infarctions.

SUMMARY

1. One hundred and forty cases are reported, sixty of whom were treated with dicumarol and eighty were treated without this drug.

2. Mortality rate was 26.25% in the control group and 15% in the group treated with dicumarol.

3. There were eight patients in the control group who had thromboembolic phenomena, six of whom died.

4. Only one instance of extension of the infarction was found in the dicumarol group. This was not considered to be a dicumarol failure since the prothrombin time was not within the therapeutic range.

TABLE VI
ANALYSIS OF DEATHS

Case	Age	Sex	Dicumarol Level	Time Weeks	Evidence of Thromboembolism	Autopsy	Cause of Deaths
W.M.	71	M	Satisfactory	6	No	Yes—Perforation of Septum	Congestive Failure
E.B.	61	M	Satisfactory	1	No	No	Congestive Failure
G.T.	65	M	Satisfactory	1	No	Yes	Congestive Failure
G.S.	60	M	Not Satisfactory	1	No	No	Unknown
W.W.	75	M	Satisfactory	1	No	No	Shock, Cheyne-Stokes Unknown
W.E.	80	M	Satisfactory	2	No	Yes	Unknown
A.B.	69	F	Satisfactory	3	No	No	Congestive Failure
A.G.	73	F	Not Satisfactory	2	No	No	Congestive Failure
E.L.	61	M	Satisfactory	2	No	Yes	Congestive Failure

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CONCLUSION

From this series and others, it appears evident that dicumarol is a valuable adjunct in the management of acute myocardial infarction.

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DISCUSSION

DR. PAUL D. CAMP, Richmond: I greatly enjoyed reviewing Dr. Beckwith's very excellent paper and also his very good presentation today. He has covered the subject so thoroughly that there is little, if anything, to add. I shall try to emphasize some points that he could not dwell on in detail because of the limitation of time.

Heparin was used as an anticoagulant before dicumarol, but, due to disadvantages Dr. Beckwith mentioned, its use in coronary thrombosis has been rather limited. Probably with the further development and refinement of Pitkin's menstruum or some similar substance, depo-heparin (Upjohn) there will be certain cases in which it would be wise to start immediately on heparin, in order to gain advantage of its rapid action, and then switch over to or start simultaneously on dicumarol, stopping the heparin as the dicumarol action takes effect; such cases might be those brought into the hospital showing definite evidence of an extension of a myocardial infarct, and/or cases of myocardial infarction brought into the hospital presenting definite evidence of additional thromboembolic phenomena.

Dicumarol, I believe, is the drug of choice in the majority of cases for reasons given by Dr. Beckwith. Furthermore, we have the benefit of knowledge gained by its use in many cases and its proven ability to lessen the total mortality in myocardial infarction.

I have, of course, used dicumarol in many cases of myocardial infarction; in fact, unless there is some contraindication and if the patient can be hospitalized, I use it practically routinely. It has certainly been my clinical impression that dicumarol therapy does reduce the mortality in myocardial infarction. I have intended doing a statistical check up on my cases and I hope to do so in the near future.

Dr. Beckwith is to be congratulated for his excellent results, and his results most certainly do compare very favorably with other published series. It is interesting to note that of the nine cases that died, six died of

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congestive failure, which again calls our attention to the severity of this complication and our inability to alter its course despite accepted methods of treatment in many cases. I should like to repeat for emphasis, that in the treated series there was no instance of thromboembolic complications while the patients were on adequate dicumarol therapy.

Finally, a word in regard to the contra-indication and complications of this type of therapy. I strongly agree with Dr. Beckwith that dicumarol should be given only in circumstances where we can get accurate daily prothrombin determinations and if the prothrombin level is initially below the desired therapeutic level, as it sometimes may be, I think it wise to withhold dicumarol until the prothrombin time rises above the therapeutic zone. I have seen one case, in consultation, in which I believe that hemorrhage was a definite complication and possibly had some effect on the patient's lethal exodus. Briefly, this patient had had a severe myocardial infarction and was being properly treated with dicumarol; the day before I saw him I believe he had a slight hemorrhage of the mucous membranes of the nose, which was thought to be traumatic as he was receiving oxygen by nasal catheter. An indwelling tube into body cavities is contra-indicated. However, the day I saw him there was a suggestion of slight hemorrhage of the lower bowel. I suggested discontinuing dicumarol but did not urge vitamin K therapy, which, in retrospect, I should have done. The next day the patient had considerably more hemorrhage from the bowel, and died later in acute pulmonary edema. Another case with chronic rheumatic heart disease, with a superimposed arteriosclerotic and hypertensive heart disease and myocardial infarction, was being treated with dicumarol and developed what was clinically a cerebral embolus and hemiplegia. I think this represented a co-incidental embolic phenomenon which is not infrequently seen in chronic rheumatic valvular disease. However, I did not have the courage to continue the dicumarol therapy, fearing that the episode perhaps represented a true hemorrhagic complication.

THE DIFFERENTIAL DIAGNOSIS OF ACUTE INFECTIOUS "IDIOPATHIC" PERICARDITIS AND MYOCARDIAL INFARCTION*

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and
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Richmond, Virginia

Bonetus in 1700 published the first correlation of severe coronary disease and serious illness, describing a case of sudden death which showed at autopsy calcified coronary arteries and probable occlusion. Adam Hammer in 1876 made the first bed-side diagnosis of coronary thrombosis proved at autopsy. However, very little advancement occurred in the ante mortem diagnosis of this disease until Herrick's classical work in 1912. Richard Lower in 1669 described a case of adhesive pericarditis with autopsy. In 1896 William Ewart described ten points of aid in the diagnosis of pericardial effusion and discussed the question of surgical treatment. Today it is generally recognized that both coronary thrombosis and acute pericarditis are common diseases, the latter occurring in about 5% of postmortem examinations, and in the acute phase in from ½ to ⅔ of the total.

Acute pericarditis is frequently a complication of acute rheumatic fever with pancarditis. Bacterial infection is frequently the cause of acute pericarditis, it being found often as a complication of the primary disease. There are three other types of acute pericarditis which are noninfectious in origin, two of these, namely the pericarditis associated with myocardial infarction and that associated with uremia are common; the other accompanying trauma is rare. In addition to the above there is another type of pericarditis in which we have been quite interested and which we wish to discuss today; namely, acute infectious "idiopathic" pericarditis, which often simulates coronary occlusion. Barnes and Burchell in 1942 first described this condition as a distinctive form of acute pericarditis of benign non-suppurative nature with pain in the chest as a frequent complaint. Massey in 1947 suggested the name "noncommittal idiopathic" pericarditis for this particular type of pericarditis.

We believe that this type of acute "idiopathic" pericarditis has in recent years perhaps been confused with coronary occlusion, for at the present time the probability that the diagnosis of coronary occlusion is made too frequently in cases having

*Read before the annual meeting of the Medical Society of Virginia at Fort Monroe, October 9-12, 1949.

chest pain, cannot be doubted. We, of course, realize that this select group of cases of pericarditis is rare when compared with the number of persons having coronary occlusion. However, since the method of treatment and the immediate and remote prognosis is so different in the two conditions, the importance of a correct differential diagnosis must be emphasized. The diagnosis of a myocardial infarction should impose a grave immediate prognosis, a prolonged convalescence and usually a marked change in the patient's life and activities. Whereas, if acute "idiopathic" pericarditis is diagnosed correctly, the immediate and remote prognosis is good, the convalescence is usually relatively short and the patient can usually return to his previous normal life.

The clinical and laboratory features of help in the differential diagnosis of these two diseases are discussed below.

Age. Barnes and Burchell report the group tended to be younger with the pericarditis than with the pure coronary occlusion. The average age of their patients who had had coronary occlusion was about 55 years as compared with 42 years of age in the group presenting acute pericarditis.

History of some preceding acute infection. Nearly all observers have mentioned the fact that there is usually a history of some acute infection preceding the episode of pericarditis and this has been our experience. Usually it is in the upper respiratory tract. Barnes and Burchell found their figures to be that 50% had a history of upper respiratory infection preceding the initial attack of pericarditis. This, of course, is not the case of patients suffering from attacks of coronary thrombosis.

Pain is one of the most dramatic factors in the picture. The pain in acute pericarditis shows certain differences from that of acute coronary thrombosis. It is generally less severe and often requires less morphine for results. Usually it is impossible on a purely descriptive basis to distinguish the cause of the pain, as terms such as "pressure" feeling, heaviness, constrictive, "crushing", and the like are used by the patient to describe the sensations produced by infarction and pericarditis. The pain of acute "idiopathic" pericarditis is frequently intermittent and there may be short premonitory paroxysms unrelated to exertion preceding the attack of severe pain which forces the patient to request medical care. As has been mentioned, the pain is usually located in the substernal or epigastric or precordial region, but in a few instances it has been localized curiously over a small precordial area well to the left of the midline. The pain rarely extends to other regions, but in some instances there may be unilateral or bilateral pain in the shoulder and hip or diffuse pain in the sides of the chest and back. As a rule this pain does not radiate. It has been noted quite often that deep breathing and rotation of the trunk or swallowing may aggravate the pain. In some instances the history indicated that the patient was most comfortable in the sitting position leaning forward. Although exertion may produce the pain, frequently it will be found to be due to flexion or twisting of the trunk and the distress may persist for hours after the exertion ceases. Long after recovery from the acute phase of the disease the patient may experience attacks of pain in the chest of variable location and duration without any reference to exertion. The pain in pericarditis is usually caused by (1) excessive fluid or (2) pleural pericardial involvement. It may radiate to the sensory area supplied by the first five thoracic segments of the spinal cord. Onset of pericardial pain in this lesion occurs from the very beginning of distress, there being no time lag in its advent as in acute myocardial infarction. In the latter, aggravation by respiration and movement does not occur until 24 to 48 hours after the initial attack. A pleural friction rub may be heard in some cases. Small amounts of pleural effusion may be present. Pericardial rubs are usual and may be heard from some period of time even though pericardial effusion may be minimal.

The type of febrile reaction is of help in the differential diagnosis. Considerable elevation of body temperature as an initial sign is usual in "idiopathic" pericarditis. Temperature elevations from 103° F. to 104° F. are observed and often persist for almost two weeks. This association of the pulse-temperature ratio with a relative bradycardia probably represents the average picture of the febrile reaction. Temperature curves in both pericarditis and myocardial infarction may be rather irregular and erratic with pericarditis maintaining the elevated level longer. Sharp febrile reactions, particularly if they respond rapidly to treatment with antibiotics, speak for an infectious origin of the pericardial reaction.

Certain differences in Roentgenological studies are interesting. Roesler points out these small quantities

of effusion present in pericarditis may not be demonstrable until 300 c.c.s or more have accumulated. Sometimes a pleural thickening may be demonstrated, although this apparently has little direct connection with the production of acute symptomatology. In some cases of pericarditis the cardiac silhouette may be increased considerably in size. There can be cardiac enlargement in acute myocardial infarction. However, such degrees of cardiac enlargement are unusual. Consequently, if a huge cardiac silhouette is observed roentgenographically it favors the diagnosis of pericarditis rather than that of coronary occlusion.

One other factor has been observed in a case at the Medical College of Virginia and that is that the fluid in pericarditis of the non-committal or "idiopathic" type has been noted to be hemorrhagic in nature, whereas that of coronary occlusion, if a pericarditis is present and an effusion develops, is usually a clear effusion.

The type of dyspnea presented by the patient is important. Interference with respiratory efforts because of respiratory pain aggravation produces a type of dyspnea most often seen in acute "idiopathic" pericarditis. An intrinsic disturbance of the respiration is not essential. Typically, a shallow, rapid, painful type of respiratory cycle is observed. If associated with primary pulmonary pathology, obviously the adventitious sounds inherent in that disease would be present. However, this is not the case in pericarditis.

The findings on examination of the heart may be of help. There may be a disturbance of the cardiac sinus mechanism. These most commonly are (1) a sinus arrhythmia and (2) paroxysmal tachycardia. Grossly irregular rhythms are not the rule. Acute "idiopathic" pericarditis presents predominantly a pericardial friction rub which is detectable several hours after the onset of the disease and remains audible for longer periods of time, even up to several weeks. This is in contradistinction to the fleeting rub associated with localized areas of infarction which makes its appearance later after the first 24 hours.

Laboratory aid is obtained mainly through the leukocyte count. A polymorphonuclear leukocytosis is common to both pericarditis and myocardial infarction. The white blood count may not infrequently be entirely normal in pericarditis. Comparative values are practically worthless in differentiating acute pericarditis from a myocardial infarction, since high white blood counts may be suggested. However, the duration of the leukocytosis tends to be longer than in myocardial infarction. The sedimentation rate is not helpful.

The electrocardiographic studies probably present the greatest single aid in the differential diagnosis. Even then acute "idiopathic" pericarditis may be erroneously diagnosed, although the only other lesion which must be excluded, perhaps, is that of hemopericardium. This must be differentiated clinically because its electrocardiogram is practically identical with that of acute pericarditis. First description of that picture was made in 1928 by Scott, Feil and Katz. Distinction electrocardiographically between acute pericarditis and coronary occlusion depends on two things: first, it implies complete familiarity with the pattern of changes in the standard and precordial leads which are produced by myocardial infarctions in various locations. Of course, it must be admitted that infarctions of unusual location or size or multiple infarctions occasionally produce electrocardiographic changes that cannot be classified. Second, it is necessary to understand the various general patterns of electrocardiographic changes that are produced by acute and subacute pericarditis. The Q1, T1, and Q3, T3 changes in anterior and posterior infarctions of the left ventricle are well known and need no comment. We recognize many variations from these patterns, which, if they occur in a serial manner after an attack of pain in the chest, will support a diagnosis of coronary occlusion. The electrocardiographic changes, described by Wood and his associates, in infarction of the lateral walls may become established in time as another characteristic pattern.

The chief electrocardiographic changes noted in acute pericarditis are RS-T segment deflections and T wave changes, without a deep, wide or abnormal Q wave, for the injury is only superficial. The RS-T segment may show an abnormal upward convexity, or may be obliquely elevated. According to Goldberger, it may show a normal downward convexity or any pattren of myocardial injury.

Usually following the return of the RS-T segment to the base line, downward symmetrical T waves develop in leads that had an elevated RS-T segment, while upward symmetrical T-waves develop in leads

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that had depressed RS-T segments. There are cases in which the abnormal T-wave does not develop following return of the RS-T segment to the base line.

In generalized pericarditis there is elevation of the RS-T segments in one or more of the precordial leads, in leads AVL and AVF, while a depressed RS-T segment is seen in AVR. Likewise, there will be elevated RS-T segment in the three standard leads.

The changes mentioned in the electrocardiographic pattern are due chiefly to the injury of the superficial layers of the heart muscle. This being the case, some of the changes are very similar to those that follow myocardial infarction.

A superficial infarct can cause RS-T elevation with a normal Q wave; however, lead AVL and AVF usually show reciprocal RS-T and T changes, due to localization of the injured area, while in pericarditis these leads show not reciprocal but similar RS-T and T changes, for the injury is usually diffuse.

The magnitude of the T wave is usually longer in infarction than in pericarditis. The RS-T segment deviations of pericarditis are not so marked as in infarction, nor do they last as long.

There is quite often a pericarditis complicating a myocardial infarct. There is usually only a pattern of infarct for the area of pericarditis is localized over the surface of the injury; however, Langendorf points out there may be a diffuse pericarditis of sero-fibrinous type following myocardial infarct.

Should this be the case in an anterior infarct, Goldberger states that the precordial leads and lead AVL show a QR pattern and an elevated RS-T; this is due to the fact that they face the epicordial surface of the infarct. Lead AVF has a normal QRS, but also elevated RS-T because it is facing the epicordial surface injured by the diffuse pericarditis, instead of the uncomplicated infarction pattern of depressed RS-T segment.

In a case of posterior infarct complicated by diffuse pericarditis, lead AVF shows a QR pattern and elevated RS-T, while the precordial leads and lead AVL show normal QRS but have elevated RS-T segment.

Clinically there may be a pericarditis with or without diffusion, but the electrocardiogram remains normal. However, the RS-T elevation in one or more of the precordial leads, lead AVL and AVF, with normal Q waves in these leads and a depressed RS-T in AVR, is most characteristic of acute pericarditis.

Ten cases illustrating this type of "idiopathic" pericarditis are summarized below.

CASE 1:

This patient, W.B.S., was a 52 year old white male in good health until three weeks ago when he began to note malaise and dull pain under the lower precordium. He went to bed, but felt no better. The dull pain has continued and lately there has been some soreness beneath the sternum. No fever during the first two weeks. For the past several days there has been fever, low in the morning and rising in the evening as high as 102°. There has been some cough without sputum during the past week. Substernal pain seems aggravated by cough. There has been no real pleuritic pain. Appetite has been poor and for several days the patient has had some vomiting brought on by paroxysms of coughing. Chest x-rays taken at home have been negative and EKG's have been within normal limits. White blood count has been about 12,000 with a predominance of lymphocytes and mononuclear cells. The sedimentation rate has been about 20 mm. an hour. Chief physical findings noted have been a prominent pericardial friction rub over the sternum present since the third or fourth day of the illness. Examination on admission showed the heart within normal limits to percussion, regular rhythm, sounds not remarkable, except for a to and fro friction rub over the whole heart, but loudest over the upper precordium and much less intense at the apex. No murmurs were heard. Lungs: in the left base there is dullness below the angle of the scapula with breath sounds diminished in intensity, but of slight increased pitch. Few very fine rales were heard in the left base after coughing. Tactile fremitus diminished over left base. Lungs otherwise clear.

Urinalysis on admission showed color amber, reaction acid, specific gravity 1.008, albumin 1 plus, sugar negative, acetone negative, diacetic acid negative, 1-2 w.b.c. per h.p.f., and 4-6 r.b.c. per h.p.f., and rare hyalin cast seen.

Blood count showed 3.8 red cells, 81% hemoglobin, 11,100 white cells, 73% polys, 1% eosinophils, 1% basophils, 16% lymphs, 9% monos, 1% reticulocytes. Six days later the red count was 3,980,000, hemoglobin 82%, white count 8,400, 69% polys, 24% lymphs, 7% monos. Eleven days later the red count was 4,320,000, hemoglobin 88%, white count 5,750, 75% polys, 2% eosinophils, 14% lymphs, 9% monos. Blood cultures were negative. Sedimen-

tation rate was 26 mm. in one hour. Routine agglutinations were negative. Second blood culture was negative. Electrocardiogram done on March 25, 1942 showed auricular rate of 100, ventricular rate of 100, P.R. interval .16, Q.R.S. interval .06, lead I convex ST with a negative T, lead II convex ST with a negative T, lead III deep Q, convex ST and a flat T, lead IV elevated ST and a negative T. The conclusion was convex ST with negative T changes means pericarditis.

On fluoroscopy the transverse diameter of the heart measured 6.3 cm. and the transverse of the thorax 34.4 cm. The cardiothoracic ratio was 50%. The transverse of the great vessels 6.2 cm. There was slight enlargement of the heart toward the left with predominance of the left auricle and ventricle. There was very forceful pulsation of the auricles and ventricles and a thrust like pulsation of the aortic knob upward toward the left suggesting the arteriosclerotic type. There was a moderate lower left pleural effusion well posterior extending over the left diaphragm and upward to the 8th rib near the mid axillary line, but mostly the density was adjacent to the posterior axillary line. There was no parenchymatous tuberculous lesion noted in the upper lung. There was slight mottling of the lower hyalin and trunk markings with prominence of the left interlobar pleura. Conclusion: no evidence of pericardial effusion or a definite thickening of the pericardium at this time, small left posterior pleural effusion which could well be the residual or revised pneumonia. Re-examination of the chest five days later showed less lower left pleural effusion with excellent pulsation of the auricles, ventricles and aorta and pulmonary blood vessels. Improvement was suggested with no pericardial effusion seen. Repeat examination of the chest eight days later, re-examination with posterior-anterior view, left lateral fluoroscopy showed pleural effusion, probably little less than on previous examination. Pulsation of the heart and aorta was not limited.

The patient was admitted on March 24, 1942 and on March 27, 1942 the following progress note was made: "Temperature down to practically normal yesterday, patient looks and feels better, less cough, very little precordial pain. Examination of heart reveals a pericardial friction rub which, widely audible over the whole precordium three days ago, is now heard only faintly over the 4th intercostal space just to the left of the sternum. Heart sounds elsewhere are normal. Lungs: sign of fluid in the left base still present, perhaps somewhat less than before."

March 31, 1942 there was an ENT consultation which showed the nose with the recovering stage of nasal membrane, dry and with crust formation. Pharynx and tonsils clear at this examination. There was no active treatment recommended at this time. Seen by Dr. Peter N. Pastore. The patient was discharged on April 2, 1942.

This patient has been under observation since the time of discharge and there has been no residual or recurrence of this manifestation of pericardial disease.

CASE II:

This 20 year old white female was admitted to the Medical College of Virginia Hospital with a history of being well until two weeks ago at which time she developed an upper respiratory infection. Shortly afterwards she suddenly developed an acute oppressing pain in her chest which did not increase or decrease on respirations. The pain radiated to the left side of the neck and down the left arm. There was a tachycardia and some fever. Systems review was essentially negative except for some urinary frequency and a history of occasional hematuria.

Past history: usual childhood diseases, no other serious illness, operations or injuries. Family history: mother and father both died very young, father from flu in 1918, cause of mother's death was questionable. There were five brothers and sisters, all living and well. No familial disease elicited. Social history non-contributory.

Physical examination: Blood pressure 125/75, pulse 100, respirations 20, temperature 99.2°. The patient was a well developed, well nourished, white female lying in bed, appeared quite active for her condition and in no distress, except for an ache in her left arm. She was mentally alert and cooperative. Head and neck were essentially normal. The ENT were essentially normal. Chest expansion was equal and bilateral, clear to auscultation and percussion. Heart: PMI was inside the mid clavicular line at the 5th intercostal space, sounds of rather poor quality, rhythm regular, there were no murmurs. The A2 appeared greater than the P2. The abdomen was soft, moderately obese, no spasm, masses or tenderness, no organs palpable. Extremities were essentially negative. Rectal and pelvic exams were noncontributory. No lymphadenopathy noted. Urinaly-

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sis on admission showed urine to be straw colored, reaction acid, specific gravity 1.006, albumin was negative, sugar negative and acetone negative. There were 10-12 w.b.c. per h.p.f. and 0-1 r.b.c. per h.p.f. There were frequent epithelial cells seen.

Blood count showed 4,570,000 red cells, 84% hemoglobin, 7,200 white cells, 75% polys, 2% eosinophils, 21% lymphs, 2% monos. Blood count seven days later showed red count to be 4,470,000, hemoglobin 88%, white count 8,200, polys 68%, eosinophils 10%, lymphs 20%, monos 2%. Four blood cultures done respectively on 9-9, 9-13, 9-16, and 9-23 were negative.

Chest showed cardiothoracic ratio of 44%. The heart was slightly mitral in type with slight prominence of both auricles. The lungs were clear. Sed. rate on 9-11 was 27, 9-16 was 25, 9-17 was 27, 9-27 was 28. Electrocardiogram done on 9-9 showed auricular rate of 88, ventricular rate 88, rhythm regular, P.R. interval .16, Q.R.S. .06, lead I, convex ST negative T, lead II, convex ST negative T, lead III, low R convex ST negative T, lead IV, convex ST negative T. Conclusion: convex ST and T changes suggest pericarditis.

Basal metabolism done on 9-25-46 was minus 10. The patient ran a low grade fever between 99° and 99.5° until day before discharge from the hospital. Progress note made on 9-20, "EKG changes not differing a great deal from previous tracings. All would suggest acute pericarditis". Progress note on 9-27, "Patient called tonight complaining of pain in arm and pericardium. Exam revealed a very frightened and apprehensive female. The heart rate was 76, rhythm regular, sounds of good quality. The patient was reassured and there was no further trouble."

The patient was discharged on 9-30, after twenty-two days of hospitalization, temperature was normal and there was no further pain noted. The patient has had no repeats of this episode during the past four years. Present state of health good.

CASE III:

This twenty-one year old white male was well until two months before admission to the Veteran's Administration Hospital at which time he developed a "tired" feeling throughout the anterior chest. Several hours later he was awakened by crushing, severe pain beneath the sternum with radiation to both shoulders. This was relieved to a slight degree by a forward bowing of the shoulders. The patient was hospitalized twenty-four hours later and remained in the hospital for one week, gradually improving symptomatically. Following his discharge and up until the time of his second admission, he had a feeling of fullness in the anterior chest with occasional sharp shooting pains along the lower rib margin anteriorly, relieved by belching. Two weeks prior to admission he noticed a soreness across the mid back on arising in the morning. There had been no chills or fever and no less appetite although he claims to have lost fourteen pounds during the two month period of the above symptoms.

Physical examination on admission was essentially negative except for a temperature of 101 degree. The lungs were clear. The blood pressure was 115/60. There was a short, blowing systolic murmur in the pulmonic area. The rhythm was normal and no friction rubs were heard. There was slight tenderness in the right upper quadrant and the liver was barely palpable.

Laboratory data on admission showed a urinalysis, Kahn, icteric index, a total and one minute serum bilirubin, cholesterol and cholesterol esters, flocculation tests, all within normal limits. There was a moderate leukocytosis. The electrocardiogram revealed a sinus arrhythmia. In Lead I there was a moderate W-shaped QRS and a low T wave; in Lead II there was a low T wave; and Lead III showed an inverted T. CF II revealed a right side pattern and CF IV and CF V showed low T waves. A second electrocardiogram taken ten days later showed a regular rhythm, Lead I had a small R and a small S and a low T, Lead II showed a low T with a questionable diaphasic T, and there was a low inverted T in Lead III. No left sided pattern was found in the CF leads and the T waves were low.

X-ray revealed a flat plate of the abdomen that was negative; radiographic and fluoroscopic examination of the chest show the left hilum to be somewhat prominent as though the heart were pulled to the left by adhesions. Adhesions were seen between the left diaphragm and the left lungs; the middle segment of the left border of the heart was somewhat prominent and it was felt that this was probably due to the adhesions described above.

The patient's hospital course was uneventful after the second day. Daily blood pressure determinations were normal and the patient was asymptomatic. Examination of his heart and lungs by the cardiovascu-

lar service was found to be normal on several examinations. The patient was discharged on the twelfth hospital day with a diagnosis of a non-specific pericarditis.

CASE IV:

This fifty year old colored male was in good health until three weeks prior to admission at which time following a "head and chest cold" the patient developed a sudden sharp pain across his anterior chest, which was aggravated by deep inspiration and seemed to cut his breath off. This was followed by soreness which remained in the left lower anterior chest around the left costal margin. Other than this soreness the patient was relatively comfortable until three days before admission, at which time the pain in his chest became markedly worse and the patient lost his appetite. The patient gave a history of a similar episode two years ago, lasting for approximately one week and since that time he has had two or three similar attacks.

Upon initial examination the patient's temperature was 101 degree and positive findings were noted as follows: The respirations were short and somewhat rapid. There was some dullness and depressed breath sounds over the left base posteriorly and laterally. There were some crackling, inspiratory rales heard at both bases. The heart appeared enlarged to percussion with a regular rhythm and a rate of 126. The remainder of the physical examination was essentially normal.

Laboratory studies on admission showed a hemoglobin of 12.2 grams, a red blood count of 4,500,000, a white blood count of 14,500 with 84% neutrophils, 10% lymphocytes, 4% monocytes, 1% eosiniphils, 1% basophils. The sedimentation rate was 34 mm.

Chest x-ray on admission reported that the lungs were clear, the heart was greatly enlarged both to the right and left. Fluoroscopy of the heart revealed the cardiac shadow to be greatly enlarged, both to the right and left, with some posterior displacement of the esophagus. The cardiac pulsations were markedly reduced. Repeat chest x-ray twenty-four days after admission showed the lungs to be normal. X-ray and fluoroscopic examination of the heart showed the transverse cardiac diameter now to be slightly above 50%. No particular chambers seemed to be enlarged and pulsations were normal.

Electrocardiograms taken shortly after admission revealed a sinus tachycardia, left axis deviation, inverted T wave in Lead I and II. Recheck five days later showed T1 and T2 to be low. A third check was done with the use of unipolar leads with no abnormalities being noted. The patient ran a moderate degree of fever, which slowly came to normal over a period of several weeks, and the patient was discharged from the hospital thirty-four days after admission, asymptomatic.

CASE V:

This thirty-three year old white male was admitted to the hospital because of the onset of mild aching pain in the epigastrium and lower chest radiating to both shoulders three weeks previously. The pains were inconstant and not related to meal. The symptoms continued and twelve days later he passed a tarry stool. Two days following this he was awakened by a severe sharp pain in both lateral costal areas and a severe gripping pain in the epigastrium, causing him to vomit. Since that time and up until the time of admission he had noted a sharp pain in the left lower anterior chest, accentuated by deep breathing or movement.

Physical examination on admission showed the patient to have a temperature of 102 degrees. There was soreness to touch of the anterior chest. There was a systolic friction rub heard over the fifth intercostal space just to the left of the sternum. The remainder of the physical examination was essentially normal.

Laboratory studies revealed a hemoglobin of 14.7 grams, white cell count of 9,400 with 76% neutrophils, 22% lymphocytes, 2% monocytes. The sedimentation rate was thirty-four mm. X-ray of the chest revealed the heart and lungs to be within normal limits.

The electrocardiogram on admission showed the following change: a flat ST 1 with an inverted T1; a slightly elevated ST 2 and a diaphasic T2; a low voltage QRS 3 with a small T3. There was T wave inversion in CF2, 4 and 5. It was thought that the ST and T changes were compatible with acute pericardial disease, and four days later a second tracing revealed an inverted T1 and T2, a low T3 and a small Q 3, and the chest leads showed a depressed ST and CF 5, and an inverted T in CF 2, 4 and 5. The third tracing made twelve days later showed an inverted T1, T2, and T3, a small Q3, and an inverted T V4 and V5.

The patient's hospital course was essentially un-

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eventful. He ran a fever for the first two days of admission but none thereafter. He was treated with bed rest and shortly after admission became asymptomatic and remained so, being discharged fourteen days after admission.

CASE VI:

This fifty-five year old white male was well until three weeks before admission at which time he developed an upper respiratory infection and a cough, productive of a yellow sputum. Three days before admission the patient noted a severe crushing precordial pain with soreness, but no radiation. The pain became more marked on walking or attempting any form of movement. It was not affected by eating or deep inspiration. The patient's appetite became poor and he was unable to sleep.

The temperature on admission was 102 degrees. The pharynx was injected. The heart sounds appeared to be somewhat distant in quality. P2 was greater than A2 and there was a to and fro grating rub heard over the sternum and to the left of the sternum in the third intercostal space. The blood pressure was 124/90.

White count on admission was 6,500 with 79% neutrophils. The sedimentation rate was thirty-five mm.

Electrocardiogram on admission showed a questionable low T1, 2 and 4.

X-ray showed an increase in the transverse diameter of both sides of the heart. The anterior mediastinum was widened. There was no evidence of a pericardial effusion.

The patient remained in the hospital for fifty-two days. During that time he developed T wave inversion in Leads I, II, and IV on the fourth hospital day, and these subsequently returned to normal during his hospitalization.

On the sixth hospital day there was some left ventricular enlargement noted by x-ray. On the thirty-first hospital day fluoroscopic examination of the heart was normal. The patient gradually improved and at the end of the fifty-two days was discharged, asymptomatic.

CASE VII:

Ten days prior to admission this forty-nine year old white male developed a dull, non-migratory pain in the left shoulder while sitting in the barber chair. The pain penetrated inward from the outer portion of the shoulder into the clavicle and continued for twenty-four hours, being associated with drowsiness. The pain then changed in nature becoming a sticking type radiating from the left scapula to the right scapula and was severe enough to confine him to bed. The following day an electrocardiogram was taken and reported to be normal and with the use of sedation he became fairly comfortable although he developed a moderate degree of anorexia. Nine days following the initial attack he developed a fever of 101 degrees and the pain became a dull persistent sticking type just beneath the clavicle bilaterally, but more severe on the left and accentuated by deep inspiration. He was then hospitalized at a private institution for six days, during which time he received anticoagulant therapy, penicillin and sedation. He showed no improvement during this time and upon request was transferred to the Veteran's Administration Hospital.

On admission he was found to have a fever of 99 degrees, which went up to 103 on the second hospital day, but by the fifth hospital day it had returned to normal. The pulse varied between 80 and 100, and respirations were between 20 and 25. The blood pressure was within normal limits.

The white blood count showed 10,800 white cells with 80% polys; on the third hospital day it was 10,550 with 86% polys and on the eighth hospital day it was 9,700. On the day of discharge the patient had a normal white count with a normal differential. Sedimentation rate on admission was twenty, gradually increased to thirty-six, and by the time of discharge had returned to normal.

The electrocardiogram showed an increased amplitude of the QRS complexes and inversion of T1, T2, CF 4 and 5. These changes persisted until discharge.

X-ray of the chest was interpreted as suggesting a pericardial effusion on the first day. Ten days later x-ray and fluoroscopic examination suggested some left ventricular enlargement though no effusion was found at this time.

At no time during the hospital stay were any abnormal physical findings noted in the lungs or the heart.

The patient was discharged from the hospital on the twelfth hospital day, asymptomatic; however, during this time his electrocardiogram had not returned to normal.

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CASE VIII:

This 50 year old colored female had been well until approximately twenty days before hospital admission at which time she began to have upper respiratory infection with much cough. A week after the onset of these symptoms the patient noted a severe aching pain involving the entire left chest and at times, the back of the neck. Respiratory movement accentuated the pain. A week after onset she was seen by her local physician who noted that she had a fair degree of breathlessness. The pain seemed to be of a moderate, intermittent nature until the day of hospital admission, at which time she experienced a severe attack of left chest pain which was of an aching character, and it did not radiate but remained chiefly in the precordial area; it was constant and not paroxysmal. This seemed to be somewhat relieved by rest.

Physical examination on admission revealed a blood pressure of 110/90, a pulse of 80, respiration of 26 and temperature 100.2° rectally. The lungs were of normal resonance; the breath sounds appeared louder and more distinct on the left, anteriorly, with an occasional moist rale over the left base. The heart was not enlarged. The cardiac sounds were of good force and regular; no murmurs were heard. There was a coarse grating sound to the right of the sternum at the third intercostal space; it was very difficult to time. Extremities: There was a 1 plus pitting oedema of the ankles.

The day after admission the patient persisted with a definite friction rub just to the left of the sternum in the third intercostal space. The electrocardiogram showed an elevated ST1, ST V4 and ST V5. There were no Q waves. At this time the clinical picture was somewhat suggestive of coronary occlusion; however, pericarditis could not be ruled out. On the twelfth hospital day the patient experienced knife like, sticking pains just below the left breast; a cough developed with a feeling of substernal pressure.

A chest plate at this time revealed a type of leather bottle heart which on fluoroscopic examination seemed to be a pericardial effusion. The friction rub had disappeared at this time and a pericardial tap was attempted. Grossly bloody fluid was obtained, dark in color. The fourteenth hospital day a second pericardial tap was done, with 10 CC. of bloody fluid removed and a small amount of air injected.

At this time there was an elevated ST in leads I and II and VR4 and 5, which were indicative of pericarditis. However, in VR4 there was a small Q and in VR5 there was a small Q which made it difficult to rule out absolutely, an associated myocardial infarction. However, the character of the pericardial fluid made this very doubtful. The twenty-second hospital day the patient continued to complain of some precordial dull pain but there was no change in heart sounds and no rub was heard. On the thirty-second hospital day the patient was completely free of symptoms, the heart sounds were normal. At that time there were abnormal T changes in all the leads of her electrocardiogram, of an indeterminate nature.

A moderate leukocytosis up to 13 thousand remained until the nineteenth hospital day.

The patient was last seen five months after hospital discharge; she was asymptomatic, the electrocardiogram had returned to normal and the patient was performing her usual duties.

It was our impression that this was a case of nonspecific idiopathic pericarditis. Cultures done on the fluid removed from the pericardial space were negative for any growth and on smears no organisms could be found.

CASE IX:

This 45 year old white male was admitted to the hospital following an attack of "indigestion" occurring shortly after the evening meal. That afternoon, the patient suddenly developed a "squeezing" type of precordial pain, which gradually subsided. Following his attack after supper, he was seen by his local physician who discovered a peri-cardial friction rub on examination and recommended hospitalization.

Seven years previously, the patient was diagnosed as having a coronary occlusion, and was treated at home with six weeks bed rest. From that time until the onset of the present illness, the patient has had no cardiovascular symptoms.

Physical examination on admission revealed a well developed, well nourished, white male, in no acute distress.

Blood pressure 120/70, pulse 80, respiration 18, temperature 99.6.

The remainder of the physical examination was entirely negative, except for a friction rub heard best at the apex of the heart.

Laboratory findings on admission were within nor-

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mal limits except for a white count of 10,400.

X-ray of the chest was reported to be within normal limits.

Electrocardiogram on admission showed only low T waves in Leads I and VI.

Hospital course: The patient was treated symptomatically with sedation, vasodilators and analgesics, as necessary. During his hospitalization, the pulse and blood pressure did not vary to any remarkable degree. Repeated sedimentation rates and white counts were within normal limits, all during hospitalization. He developed, however, T wave inversion in AVL, V2, and V3, which persisted throughout his hospital stay.

Two days after admission, the patient became asymptomatic and the friction rub heard on admission disappeared. He remained asymptomatic during the remainder of the hospitalization of 30 days.

CASE X:

This 41 year old white male was well until ten days prior to examination, at which time he developed an upper respiratory infection with nasal congestion and an intermittent, but persistent, cough. The symptoms were not severe enough to cause the patient to cease his work; however, on the day of examination he began to feel a tightness in the center of his chest. He stopped his usual work early and attempted to return to his home. The central pain became more severe and the patient began to experience dyspnea; he was unable to take a deep breath for it increased the pain. There was no radiation of the pain, it seemed to localize substernally.

On physical examination his temperature was found to be 100° orally, his blood pressure 128/76 and pulse 98. The chest was clear as to auscultation and percussion. The heart sounds were distant. There were no friction rubs heard.

During the patient's hospital course he ran a low grade oral temperature and continued to experience substernal pain without any radiation. Movement seemed to make the pain more severe and deep breathing definitely seemed to affect the pain. On the sixth hospital day a loud pericardial friction rub was found in the fourth intercostal space at the parasternal line. The chest was clear. The electrocardiogram at this time showed a small Q2, a slightly elevated ST2 and an upright T2; there was a small Q3, a low T3 and an elevated ST3.

The heart shadow appeared definitely enlarged and the mediastinal shadow broad, suggesting some fluid in the pericardial space in addition to an enlarged heart. The lungs were clear. A pericardial tap was done and no fluid was recovered; this procedure was repeated with moderate saline washing being done and the material collected for culture. There was no great amount of fluid obtained at this time.

Pericardial friction rubs persisted until the 19th hospital day. The patient was being treated with penicillin and the temperature fell by crisis on the 14th hospital day. Following the 19th hospital day, the patient continued to improve with only occasional substernal pain of the non-radiating type, frequently affected by respiration. On the 28th hospital day the patient was allowed up and about, with no discomfort, and was discharged from the hospital on the 31st hospital day, feeling well and with only occasional discomfort over the pericardium on deep inspiration.

The culture of the pericardial washing revealed a hemolytic streptococci present in pure culture. This was confirmed by a second report, a culture of sediment showing a hemolytic streptococci present.

In view of the clinical picture presented, supported by the electrocardiographic changes, it was impressioned that this patient had a streptococcic pericarditis, secondary to a severe upper respiratory infection.

This patient was last seen four months after hospital discharge. The electrocardiogram was essentially normal. All laboratory studies were normal. The patient was doing full time office work.

COMMENT: Nine cases of acute infectious idiopathic pericarditis have been presented. The tenth case represents the same clinical entity, however, due to unusual circumstances, the causative agent was found. We believe if such procedures were carried out more often the term, "idiopathic", would have to be used less often. However, we certainly do not recommend routine pericardial taps in such cases.

We wish to express our thanks to Dr. Louis Calisch for his assistance in summarizing some of the cases.

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Professional Building.

DR. WELLFORD REED, Richmond: I want to thank Dr. Camp and Dr. Clarke for bringing to our attention this very important problem in differential diagnosis.

In October, 1948, Logue and Wendkos, in the *American Heart Journal,* analyzed 40 cases of pericarditis and found 15 of them non-specific and 15 associated with rheumatic fever. The rest were tuberculous, associated with disseminated lupus, rheumatoid arthritis, trauma or neoplasms. This is important, because from the onset we see that the large majority of cases of pericarditis are non-specific, and it is possible that we miss many cases associated with upper respiratory infections. I believe it is much more difficult to make this diagnosis than that of myocardial infarction.

Regarding pain, this is usually due to involvement of the pleura or the diaphragmatic reflection of the pericardium with reference of the discomfort to the left shoulder or neck. It is of interest that some cases show relief of pain with increased accumulation of pericardial fluid. Some have a crushing type of pain, similar to that which occurs in coronary occlusion.

There is a much more difficult type of case to diagnose, which presents the typical substernal pain; but the patient has no fever, the pain subsides rather promptly, there is no elevation of leukocytes, and the electrocardiogram shows only inversion of T1 and T4 without QRS changes. These individuals usually recover so we have no post mortem studies to parallel our clinical studies and are not quite sure of what is going on within the heart structure.

As Dr. Camp stated, unless we make frequent diagnostic taps we shall diagnose an increasing number of cases as idiopathic pericarditis. On the other hand, where there is little fluid, we shouldn't risk making a bloody tap, which would confuse the picture. In my experience bloody fluid is more frequently associated with tuberculosis or malignancy. In both conditions fluid will accumulate very rapidly. This is not the case in non-specific pericarditis, when once the fluid is removed the patient usually begins to improve. We have seen one case in which the patient was relieved by the administration of aspirin only; the temperature came down, and the pain, which had been rather severe, was relieved in 24 hours. There are some things which make us feel this may be a rheumatic-fever type of disease.

Two cases seen within the last two years emphasize a little different slant on the differential diagnosis; that is, trying to differentiate pericardial disease from congestive heart failure. We saw two cases over sixty years of age, both women, and both of whom came to the hospital complaining of shortness of breath. They had abnormal electrocardiograms, neither of any specific pattern. While we were puzzling as to what it meant, a friction rub came to the rescue in both cases, and we made more extensive studies and found that both were suffering from pericarditis and had a previous history of upper respiratory infection. One thing that puzzled us was the fever, which we could not explain on the basis of heart failure alone. The conclusion from the X-ray report was that there was marked hypertrophy of the heart with passive congestion and a small amount of fluid. We removed the fluid from the left pleura, we thought; and an X-ray taken about four days later showed the heart shadow smaller, and no evidence of pleural effusion. Then we concluded that this was a pericarditis and not congestive heart failure. The second case had a similar report, which worried us as well as the X-ray man. The heart shadow was quite large. When a friction rub came along seventeen days later, we decided we were probably dealing with a pericardial effusion. Later she began to cough up a lot of sputum just loaded with acid-fast bacilli. Her effusion disappeared spontaneously before streptomycin therapy was started and we felt that she probably had a non-specific pericarditis in spite of the laboratory findings.

CHAIRMAN OUTLAND: The general discussion of these three papers will be opened by Dr. Oscar Clarke, who is co-author with Dr. Camp of the third paper.

DR. OSCAR CLARKE, Richmond: More than in the manner of discussion I wanted to re-emphasize the point Dr. Camp made as to how essential the electrocardiographic opinion is in making the diagnosis between idiopathic or acute infectious pericarditis and coronary occlusion. This is rather technical, and I hope you will bear with me as I read it. (Dr. Clarke read several pages from Dr. Camp's paper.)

CHAIRMAN OUTLAND: The floor is open now for general discussion of these three papers.

A MEMBER: Mr. Chairman, I should like to ask Dr. Porter whether the preference is for whole blood or plasma in the type of shock he discussed.

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A MEMBER: I should like to ask whether, after the administration of blood in coronary shock, it might be necessary to institute bleeding, because actually there has been no blood loss. When the patient comes out of shock is it necessary to bleed him?

CHAIRMAN OUTLAND: Are there other questions or discussions?

DR. —— MOSELEY, ——, Florida: This has certainly been a most excellent symposium, one from which I think everyone has learned something and one which puts us on our toes and will make us try to do better.

I have been using intravenous fluids for ten or fifteen years in what we might call dying cases. Dr. Horsley felt that you could not hurt a patient nor his heart if he were in shock and you gave fluid; and his opinion was this—to give it, and give it rapidly, and stop at the right time. Now, that is the question—when to give it, how much to give, and when to stop. Those are three $64 questions.

The point comes up in this way. Suppose you are in the hospital and you have a patient that has had a heart attack in one or the other end of the hospital. When you go there the patient is in shock and you become excited, maybe or maybe not. You had better not, because it is hard on the patient if you do. You look the patient over; the nurses are running; alarms are sounded; and maybe you become a little alarmed. The best thing to do is to walk up to the other end of the hall. By the time you return the patient is either better or coming out of shock or maybe is dead. Look at him again and go away. Then maybe on your third look at him the pulse is back and his color is better. That is perhaps not very scientific medicine.

Then you decide to give intravenous fluids. The room is cold; the patient is cold; you do not know how to find a vein. Well, maybe you have the surgeon cut down on the vein. Give that fluid and give it fast, and when the pulse comes back stop the fluid. Give no more fluid than necessary to maintain the pulse with the needle in the vein, a large one if you can get it in. Then observe the patient again. The more you observe these people the better judgment you will have.

It has happened twice in the last two weeks that technicians have gone to get blood in people that had no veins. In one case of shock they could not get a vein. This is something everybody knows but that lots of times they do not remember. It is simply this; put a hot-water bottle on the arm you are going to use and let it stay there a while, or put on several, maybe. Then go back a little later and pull the arm down and you will see the vein. The vein is rather shy in these people; it seems to run away. Then irritate the skin. I believe it is a reflex mechanism. But if you have that arm warm you will find the vein and be able to get in the fluid, and in this way I believe you will be able to save more coronaries than in any other way I know.

DR. E. E. HADDOCK, Richmond: Inasmuch as the tenth case Dr. Camp reported on a while ago was referred to him by me, I have a few remarks I should like to make about it, which may be of more value to general practitioners than to internists. The first is as to the matter of making the diagnosis. In rural Districts hospitals are not always available and electrocardiographic machines are not available, and it is very difficult oftentimes to make a diagnosis. I have to take off my hat to the man who made the diagnosis. It happened on an afternoon when I was off, and the family called a C. and P. doctor. He happened to be a young internist, Dr. Adney Sutphin, of Richmond. He took his portable machine with him up to the man's home, and made the tracing right there and told the family it was pericarditis.

The proper diagnosis is important particularly because of the long-range advice you want to give the patient. If this man had had coronary occlusion with myocarditis it would have changed his future life entirely. This man's job is that of field engineer for the telephone company, and in field work he has to go out with crews and go down valleys and up hills and so forth. If he had had myocarditis it would have changed his life entirely.

As Dr. Camp said, many cases of idiopathic pericarditis would not have been labeled so if taps had been made on them. But since the patients get along all right we maybe save the patient a lot of psychic trauma by not doing it.

A MEMBER: I should like to ask if Dr. Beckwith would care to comment on Linck's (?) suggestion that we use large doses of vitamin C in conjunction with dicumarol therapy.

DR. BECHWITH, closing the discussion on his part: First I want to thank Dr. Camp for his discussion.

We have not had any experience at all in the use of vitamin C with dicumarol. I understand it does enhance the value of dicumarol, but that is all I know about it.

I also want to state that I enjoyed the other two papers in this symposium very much. We have recently had a few cases of pericarditis, and I want to emphasize the differential diagnosis between pericarditis and myocardial infarction. It is often very difficult to make.

CHAIRMAN OUTLAND: Dr. Porter, will you close the discussion on your paper?

DR. PORTER, closing the discussion on his part: I appreciate very much the discussion of my paper by Dr. Beckwith and the comments made by other physicians. I enjoyed Dr. Beckwith's paper on anticoagulants very much. Because he covered the subject in his paper and because of time limitations, I said very little about anticoagulants. I would like to mention, however, that in our series of cases no embolic phenomena occurred in those patients having adequate amounts of anticoagulants and that the ten cases of embolus noted occurred in patients who had no or inadequate anticoagulant therapy.

I should like to comment on the point Dr. Camp brought out on the use of longer-acting heparin, as, for example, the depo-heparin now available. When dicumarol is used, the patients must be followed closely by tests of the prothrombin activity. This is a long and complicated test which can be carried out only in a hospital laboratory. For this reason only those patients who are in a hospital

could be treated in the past with anticoagulant therapy. With the use of prolonged-acting heparin, it seems probable that patients can be treated with this anticoagulant in the home. This therapy is managed by means of coagulation time determinations, which is a relatively simple procedure and can be done by the physician in the home.

As to the danger of giving too much blood, we are of the opinion that that is a very real hazard and should certainly be avoided. We watch the patient very closely when giving blood or plasma and if there is any sign of embarrassment to the heart, this is discontinued. So far we have had no untoward reactions from such therapy, principally because we are very careful about giving it. The choice of blood or plasma depends upon the hematocrit. If the hematocrit shows hemoconcentration, we use plasma; otherwise we use blood.

I would like to comment on Dr. Chapman's remarks on when to use blood or plasma transfusions. We do it routinely when the patients are in shock and continue until their blood pressure has been raised about shock levels. It may be that in the future such therapy will be given even though the blood pressure does not approach shock levels. If a patient's coronary circulation is accustomed to a blood pressure of 140 or 160 and his pressure drops 20 mm., it is reasonable to assume that the coronary circulation is not as adequate due to the lowering of the effective head of pressure at the coronary orifices. There may come a time when we will give a transfusion to a patient whose blood pressure has dropped only 20 mm. of mercury in order to maintain an adequate coronary circulation with a consequent improvement in the collateral circulation and, perhaps, a reduction in the size of the area of infarction.

DR. CAMP, closing the discussion on his part: I want to thank Dr. Reed and Dr. Clarke and Dr. Haddock for their discussion of my paper.

There is one point I want to add to Dr. Porter's paper. I think it is quite important to prevent shock and to treat it immediately when it does occur. I think in a number of cases plasma followed by saline will increase blood volume more than plasma alone. Certainly I have learned to fear the severe renal failure which comes on and the resulting uremia more than almost anything else in acute coronary occlusion. Maybe I have just had a run recently of bad cases. I do not know.

As to the result of putting fluid in cardiacs, I think if people are in shock and there is any tendency to congestive failure it is better to give the fluids by hypodermoclysis and give five per cent glucose in plain water rather than to give large amounts of saline intravenously, thus perhaps overloading the circulation and producing edema.

A MEMBER: Mr. Chairman, may I make a request? This is one of the most important symposiums I have ever seen, being on a subject in which all doctors are interested and one which tops the list of causes of death. I wonder if this group of papers, along with the comments, could be published together, even in the newspapers, so we could have it and study it. I have listened to it but could not get as much out of it as I wished. There are many excellent suggestions in the papers, and I think they will save lives if we can have them.

CHAIRMAN OUTLAND: I think you might relay that suggestion to the Program Committee. All the papers, I am sure, along with the comments, will be published in the official journal of the Medical Society—the VIRGINIA MEDICAL MONTHLY.

Aureomycin Reduces Childbirth Infection Possibilities.

According to a study on the use of the antibiotic in obstetric patients presented by Dr. Joseph A. Guilbeau, Jr., Dr. Emanuel B. Schoenbach, Isabelle G. Schaub, A.B., and Doris V. Latham, A.B., of the Johns Hopkins School of Medicine and Johns Hopkins Hospital, Baltimore, in June 10 *J.A.M.A.*, aureomycin is effective in lowering the possibilities of infection following childbirth.

The normal uterus after birth contains a wide variety of bacteria which is potentially disease producing. Such infection may result in irreparable damage which can jeopardize future childbearing, the report points out.

Aureomycin hydrochloride was administered to 109 patients before delivery. Only 13 (11.9%) showed positive cultures two to three days after giving birth. In a control series of 24 patients who had uncomplicated, normal deliveries, positive cultures were reported in 18 (75%) cases.

The researchers also treated a number of acute childbirth infections during the study. They report: "Aureomycin proved effective in various obstetric infections. Patients with acute and chronic infections of the urinary tract treated during pregnancy responded satisfactorily to therapy, although several relapsed when treatment was discontinued.

"Aureomycin is a desirable chemotherapeutic agent because it is effective after oral administration, it possesses a wide range of antibacterial activity and it is unassociated with serious toxic manifestations."

The report also points out that the antibiotic has the ability to reach the infant by way of the maternal blood stream in high therapeutic concentration. This, they say, is a desirable property.

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PAIN MECHANISMS ASSOCIATED WITH HEADACHE*

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The purpose of this discussion is not to consider the various and many causes of headache but one of considering the specific pain mechanisms that are involved in headaches and pain about the head structures.

It has been estimated that only about five per cent of all cases of headache are actually due to intracranial disease. The remainder are due to disturbances arising from such conditions as neuralgias, vasomotor and circulatory changes, emotional factors and reflex headache from structures outside the cranial cavity. Of these, most workers feel that vasomotor or neurocirculatory disturbances from emotional factors are largely responsible for the complaint of headache.

Much of our understanding of the headache problem has been due to the work of Wolff, Ray, Pfeiffer and their coworkers. Their work has given us much understanding and helpful information to the benefit of both the therapist and the patient. Riley, Brickner, Von Storch, Lennox, Horton and others have also opened up a number of avenues in the treatment of headache, particularly Horton whose interest in histamine sensitivity has done much for the relief of certain types of headache.

When we consider the problem of headaches, head pains and neuralgias, we attempt to link them with discomforts arising from local conditions about the areas. Most often it is difficult to uncover a specific local lesion. On the other hand, understanding some of the mechanisms of production of pain, the points of origin of pain, knowing what structures are sensitive or insensitive to pain and appreciating the areas of referred pains, gives us a much more satisfactory approach and analysis of the problem of diagnosis and treatment.

Knowledge of pain sensitivity of cranial and extra cranial structures is important. The scalp is sensitive to all forms of stimulation just as the skin is elsewhere. The galea in the region around blood vessels is sensitive to pain only. Fascia and muscles are sensitive to pain. The periosteum is only mildly sensitive to pain but in the region of the temporal, occipital and frontal areas it is the most sensitive. The cranial bone itself is insensitive and no pain fibers are distributed to the diploe and emissary veins. On the other hand all of the arteries of the scalp are sensitive to pain, the veins being largely insensitive. Constriction of a scalp artery does not cause pain but stretching in either a lateral or longitudinal manner will cause pain and even nausea and vomiting. Pain arising from this source is often of a throbbing character due to the stretching and relaxation accompanying pulsation. Pressure over the common scalp arteries—superficial temporal, occipital, post auricular, supraorbital, frontal areas and external carotid arteries—sufficient to interfere with circulation over the areas applied by them is a valuable method of differentiating the mechanism of the pain. Deep pressure over these vessels, usually accompanied by nerves, duplicates the pain, whereas moderate pressure and reduction of blood flow through the vessels will relieve the pain. Frequently there is in the history of these cases an observation by the patient that pressure around the head or over the area of complaint results in comfort and is a means of relief.

The main arteries of the dura are all very sensitive to stretching or distention. In migraine, fever, hypertension, and after histamine or from emotional stresses, distention is present and often relieved by constricting ability of ergotamine. Practically all headaches arising from intracranial arteries arise from mechanical alterations of artery tonus. Injecting the arteries with local anesthetic, ligating or severing the arteries will eliminate pain when these structures are stimulated after such procedures. Pain fibers apparently accompany these vessels and are distributed with the arteries. In the area of supply by the anterior meningeal area to the anterior fossa, alterations of tonus of the muscle of the artery wall by stretch, distention or pressure will cause pain in the forehead and eye on the same side. The posterior meningeal arteries are distributed to the under surface of the tentorium and the meninges below the tentorium. Pain from these structures is referred to the region of the occiput. The middle meningeal artery supplies all of the dura above the tentorium except for the floor of the anterior fossa and the

*Read before the annual meeting of the Medical Society of Virginia at Fort Monroe, October 9-12, 1949.

dura over the tip of the frontal lobes. Pain arising from the middle meningeal artery causes pain referral deep in the temporal-parietal region and behind the homolateral eye. The small branches of the middle meningeal differ from those of the anterior and posterior meningeals in that pain fibers accompany the small branches of the middle meningeal to the extreme terminations and pain may remain at the point of stimulation and not be referred.

The dura apart from the pain sensitive fibers along an artery is insensitive to pain except at the base where there is considerable sensitivity. In the anterior fossa the dura is sensitive and causes pain behind or about the homolateral eye. The dura of the middle cerebral fossa is quite insensitive but the posterior fossa dura is very sensitive along the floor and along the margins of the occipital and transverse sinuses. Pain is referred from these regions to the area behind the homolateral ear and the back of the head.

There is very little sensitivity of the dura of the falx cerebri but the superior sagittal sinus is sensitive and refers pain to the homolateral side and homolateral eye. The superior surface of the tentorium is sensitive and pain is referred to the homolateral forehead and eye region. Pressure from the under surface of the tentorium will cause pain referral to the same region.

The main veins near the point of their entry into the transverse and superior petrosal sinus, under the surface of the temporal lobe, are sensitive and pain is referred to the temporal region. The occipital sinus is very sensitive with pain referred to the region behind the left ear and low in the back of the head and neck region. Local injection of the upper three cervical nerves will relieve pain from these structures.

In the substance of the brain itself, the blood vessels are insensitive except for the basal blood vessels. Pressure, distention or stretch of these basal vessels cause deep, dull, diffuse headache, usually of a pulsating character and the headache is frequently accompanied by nausea. The intracranial segment of the internal carotid artery will refer pain behind the eye and low temporal region whereas the basal portion of the anterior cerebral will cause pain behind and above the homolateral eye. The vertebral artery refers pain to the occipital and suboccipital regions and the posterior inferior cerebellar artery, one that commonly thromboses, causes pain in the same region.

The actual neural pathways of the pain sensitive structures are divided according to location in regard to the tentorium. The fifth cranial nerve supplies the superior surface of the tentorium and all of the structures lying above it while the structures lying on the under surface of the tentorium or below it are supplied by the 9th, 10th and to a lesser extent the 11th, 12th and upper cervical nerves.

The pathological physiology of headache is based on disturbances of the hydrodynamic mechanisms within the head. The pain-sensitive structures of the meninges, tentorium, falx, and those distributed with the cranial vessels can be interfered with mechanically by tumors, abscesses, toxins, states of vasodilation, vasoconstriction or compression. Increased pulsations and blood excursions occur in migraine and in hypertension. Headache with increased or decreased intracranial pressure comes from traction or displacement of pain sensitive structures and does not depend upon generalized increased intracranial pressure. Traction on veins emptying into the sinuses and traction on the middle meningeal artery by extradural or subdural hematomas or abscesses accounts for headache in such conditions rather than increase of pressure. Displacement and traction on the large basal arteries cause generalized types of headache, just as inflammations at the base of the brain and over the convexity of the brain.

Cranial nerve neuralgias are frequently causes of headache and head pain. The most frequent neuralgias are those associated with the trigeminal nerve. Other neuralgias are less common but frequently involve the occipital and great occipital nerves.

Many facial neuralgias do not follow the patterns of the cranial nerve distributions. Neuralgias of the facial, glossopharyngeal, vidian, sphenopalatine and geniculate nerves are less common but vasomotor disturbances of the vessels of these nerves are important factors in atypical neuralgias and facial discomforts. Many individuals suffering from these painful afflictions show evidences of cardiovascular disorders, hypertension, arteriosclerosis or tremendous arterial lability and many have migraine or a family history of migraine. Many also have neuralgic pains in the brachial and lumbosacral plexuses. Atypical neuralgias are seen more commonly in women, are often bilateral and frequently follow

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vascular patterns rather than nerve distributions. Many of these have associated sympathetic disturbances with them such as lacrimation, edema, photophobia, flushing or blanching.

One of the very common forms of headache is that caused by hypertonicity and spasm of the neck muscles. Pain in the head, neck, shoulder, chest or precordium is frequent in cervical arthritis and is aggravated by head and neck movements, coughing, straining or sneezing. Early morning occipital headaches and posterior neck aches are often due to this cause and is very common. Occupations such as bookkeeping, typing and machine operators frequently have complaints arising from cervical spinal arthritic structures. Indurative myalgia, rheumatoid myositis, fibrositis and neuritis also can give similar headache. Asymmetries of the cervical spinal condyles and displaced or subluxated articulations are other causes for such complaints.

Headaches may be referred from a thoracic or abdominal viscera to the superficial tissues of the head through the relation of the trigeminal and the vagus. Three cases seen recently had pain referred to neck and head structures from stomach ulcers and disappeared with successful treatment of the ulcers. In many individuals a common cause of headache is eye-strain probably from reflex nervous mechanisms.

Particular attention is called to the sources of regional headache from intracranial structures. From the illustrations given there are specific areas or regions to which pain is referred about the head. These are the region of the eye and forehead, the temporal and parietal area, the occipital and suboccipital areas. Sources of these regional headaches can be grouped together into the following:

Pain in the eye and forehead region can arise from the anterior meningeal artery, dura of anterior fossa, superior surface of tentorium, transverse, straight and sagittal sinuses, Sylvanian vein, intracranial portion of internal carotid artery, vessels of circle of Willis and proximal portion of larger cerebral branches and the ophthalmic division of the trigeminal nerve.

Temporal region pain is referred from the superficial temporal artery, the middle meningeal arteries, and inferior cerebral veins of the temporal lobe.

Parietal and vertex region pain is referred from the anterior portion of the sagittal sinus and tributary veins.

Pain in the ear is referred from the internal auditory artery and dura surrounding the internal auditory meatus.

Occipital, suboccipital and post-auricular pain is referred from the undersurface of the straight and transverse sinuses, walls of occipital sinus, sigmoid sinus and adjacent dura, pontine and internal auditory artery, dura of the floor of posterior fossa and 9th and 10th cranial nerves.

Occipital and upper cervical pain is referred from posterior meningeal arteries, inferior cerebellar arteries, vertebral and basilar arteries, intracranial portions of the 11th and 12th nerves, 2nd and 3rd cervical nerves.

Pain may be referred to specific regions of the head from sites of origin in other head structures. Pain from teeth can be referred to areas supplied by the fifth or trigeminal nerve. Nasal structures and sinuses likewise can refer pain to head areas, notably frontal, temporal, vertex and also the suboccipital region and neck regions. Deep ocular structures refer pain to the offending eye and forehead region and may spread to side of head, behind the ears and down the neck.

All of these factors must be considered in analyzing a patient's complaint of headache. Careful questioning in obtaining the history will help bring out a specific region referral when the patient is apt to generalize or make light of his complaint and it may help to distinguish his complaint from the so-called nervous or emotional headache where the complaints are more apt to be bizarre and follow no definite pattern and are apt to be relieved by unusual measures.

Summary: The belief at the present time is that the majority of headaches are effected through vascular mechanisms, the pain itself being due to vasodilation, traction, stretch factors and subsequent local anemia or edema after vasoconstriction or rise in intracranial pressure. The vascular mechanisms are under nervous control and reflex action can be initiated from various parts of the body and in excitement and in the emotional states, the latter accounting for the origin of most headaches.

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DISCUSSION

DR. THOMAS N. SPESSARD, Norfolk: Dr. Klingman has presented in a concise, orderly and scientific fashion, most, if not all, that is known concerning the origin of headaches that are produced by structural intracranial change. I think that his observation that increased intracranial pressure alone does not produce headache, but that the secondary distortion of cerebral structures is the etiological mechanism, is an important contribution to our understanding of this problem. The entire paper was extremely interesting and informative.

Unfortunately, however, known organic mechanisms account for only a fractional number of headache victims. More than 90 per cent fall into the so-called functional group, whose headaches, certainly in the light of our present knowledge, are presumed to be emotionally induced. A functional reaction, of which headache is the most common, develops when an individual is incapable of dealing with the circumstances in his life at a given time. Continual demands beyond the personality's emotional capacity produce a somatic response, perhaps mild and transitory, often severe and lasting. Insecurity, fear, hostility, and resentment, especially if suppressed, are the four horsemen of functional disease, of which headache is the major reaction. The headaches associated with and caused by emotional tension and anxiety are, as a rule, different from the organic variety. They begin usually with what patients describe as a sensation of pressure. The scalp feels tight, as if a band were constricting the skull. They are little influenced by the position of the head, and tend to be more generalized, with some predisposition to the base of the skull. Many are slight, are absent on arising, but grow steadily worse as the day progresses, especially if excessive emotional stress or tension is demanded. Medically, these headaches show some response to ordinary analgesics, but respond more satisfactorily to sedation or environmental change.

I am sure all physicians, whatever the type of work, will agree with Dr. Klingman that the subject of headache is probably the No. 1 medical problem. To those who confronted this problem on the neuropsychiatric wards of military hospitals, headache was *the* headache. It was obvious that these individuals were projecting and converting their dislike of military service into headaches. So we, as civilians, convert our daily worries, resentments and frustrations. That it, she, him or they give us a headache is a daily used expression, all too true.

From Dr. Klingman's paper today, we can see the strides being made in understanding and coping with the mechanism of organic headache. No doubt some progress is also being made in the management of the functional headache. There is also no doubt that, as long as the individual has problems or circumstances which are difficult or impossible of solution, just so long will headaches continue as medicine's No. 1 challenge.

DR. KLINGMAN, closing the discussion: I wish to thank Dr. Spessard for his remarks emphasizing the tremendous differences between headaches of psychogenic or emotional origin. Time did not permit my entering into the explanation of origins of this kind. It is possible that these headaches may also be explained on the basis of interferences with vascular supply.

I should like to emphasize again that in obtaining histories of headaches you cannot spend too much time in trying to decide in your own mind the characteristics of the headache early in its course. Headaches in a particular region bear special consideration from the organic standpoint, and we must take into consideration the various mechanical factors that could be responsible. We do see headaches that are psychogenic or emotional in origin that express themselves in discomfort in the same regions, but usually a carefully elicited history will bring out the real cause. The persistence, though, of headache in these areas definitely warrants the consideration of an organic condition. It does not necessarily have to be a neoplasm but may be from vascular disturbances or some mechanical or hydrodynamic effects which may themselves be excited by some organic factor.

XUM

VAGINAL HYSTERECTOMY—
Indications and Advantages*

JAMES P. HENNESSY, M.D., F.A.C.S.,
New York, N. Y.

Vaginal hysterectomy was one of the very earliest major surgical procedures to be performed. Its history dates back to the second century, when Soranus, the Greek obstetrician who practiced in Alexandria and subsequently in Rome, seems to have originated the idea of removing the uterus through the vagina.

Berengaria de Carpi, early in the sixteenth century, is given the credit for the first authenticated description of the removal of the uterus through the vagina.

We shall not attempt to review the earlier literature on the subject, but in 1882, in this country, Christian Fenger[1] published a detailed description of his first case. In 1915, Charles H. Mayo[2] described an operation designed to correct the vesical deformity accompanying uterine prolapse as well as to remove the uterus. Since Mayo's paper there have been many others from all parts of the world. They have dealt mainly with the technique of the operation devised by Mayo, and, unfortunately, there has been a dearth of follow-up information upon which to base logical conclusions as to the efficiency of any one procedure.

The history of vaginal hysterectomy is one of gradual evolution through successive stages in the development of techniques to overcome several obstacles, namely: (1) to obtain proper exposure of the operative field; (2) to clarify the obscurity of anatomical detail due to a bloody field; (3) to prevent the recurrence of cystocele and enterocele. With the perfection of aseptic surgery abdominal operations have become much safer. Previously the mortality following abdominal surgery was so high that it was done only in cases of great urgency. The mortality after vaginal hysterectomy under comparable conditions is much lower than after abdominal hysterectomy.

In performing the operation three basic principles should be kept in mind. There should be adequate support for the remaining pelvic organs and the vaginal vault by the proper approximation and suturing of the ligamentous structure of the pelvis during the closure of the wound. Raw surfaces should be carefully protected, and adequate drainage should be maintained in all cases.

In my opinion, the indications for this procedure have definite limitations. The uterus must be relatively small (not larger than a three months pregnancy), accessible and movable. The vagina should be large enough to afford room for the operation. Large uterine tumors or inflammatory masses should not be removed through the vagina. This is possible, of course, although exceedingly difficult and accompanied by great trauma and profuse bleeding. If necessary, however, the vagina can be enlarged by a median episiotomy or by a Schuchardt incision.

In properly selected cases, vaginal hysterectomy is an ideal method and saves many lives; if unwisely used, however, it is extremely difficult and adds unnecessary risks to what might be an easy operation if done through an abdominal incision. Each procedure has its field of usefulness.

One of the questions most frequently asked by students is: "What are the indications and advantages of vaginal hysterectomy?" According to the procedure followed in this clinic, the indications are:

1. Procidentia of the uterus of varying degrees associated with rectocele and cystocele (the chief indication);
2. Small benign tumors of the uterus, not larger than a three months pregnancy;
3. Precancerous lesions of the cervix, including erosions, deep lacerations, ectropion or leukoplakia;
4. Persistent functional bleeding, especially in the menopausal period, not responding to hormone therapy;
5. Chronic subinvolution of the uterus.
6. Recurrent uterine polyps or those with potential malignant changes;
7. Persistent uterine leukorrhea, or chronic metritis;

*From the Department of Obstetrics and Gynecology, New York Policlinic Medical School and Hospital, New York, N. Y.

Presented at the meeting of the Clinical Society of the New York Policlinic Medical School and Hospital, May 1, 1950.

8. Stenosis of the cervix following radium therapy;

9. Carcinoma of the corpus uteri, using radium and x-ray as adjuncts, in cases where abdominal approach is contraindicated;

Its advantages are:

1. Vaginal hysterectomy is an operation associated with a minimum of shock and hemorrhage and with a low morbidity and mortality rate;

2. It is a total hysterectomy, thus qualifying for the advantages of an abdominal total hysterectomy;

3. The vaginal procedure is the method of choice in obese elderly patients and those who are poor surgical risks because of diabetes, cardiac disease, hypertension and nephritis;

4. The potentially infected cervix is removed, thus eliminating a possible focus of persistent cervical discharge and an irritative cause of cancer;

5. There is less danger of infection since the peritoneum is entered at its most dependent point where the tissues are more resistant to infection and where, should infection occur, drainage is more easily established;

6. There is less liability to ileus, due to minimal handling of the intestines, and less danger of bowel perforation. Postoperative adhesions and intestinal obstruction are minimized;

7. Diseased adnexa may be removed if necessary;

8. There is much less postoperative discomfort; nausea, vomiting and gastrointestinal disturbances are less frequent;

9. Peritonization can be accomplished as by the abdominal route;

10. Thrombophlebitis and pulmonary embolism are less common;

11. A moderately deep vagina is assured.

Vaginal hysterectomy is indicated in certain conditions and we should be familiar with these. In some hospitals it is almost a lost procedure and the operation is rarely mentioned. I believe that every gynecologist should be able to perform the operation when it is indicated and not have to resort to other procedures because of his limitations.

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116 East 68th Street.

Cortisone Side Effects Reduced by Smaller Dosages.

The development of a dosage of cortisone acetate to maintain improvement in cases of rheumatoid arthritis with a minimum occurrence of undesirable side effects is reported in the September 30 *J.A.M.A.*

Cortisone is not a cure for the disease, but its administration reverses crippling results. Its continued use is necessary in order to prevent the return of the pain and deformities which mark rheumatoid arthritis. The problem of physicians using the drug has been to prevent complications in side effects.

A report on the treatment of 42 patients is made by Dr. Edward W. Boland and Dr. Nathan E. Headley of Los Angeles. Based on preliminary studies, they said it appears that some severe cases and most less severe cases may be kept under adequate clinical control for long periods, and with relative safety, with smaller maintenance doses ranging from 32 to 65 mg. a day provided larger doses to suppress the disease are used initially.

"Comparatively few unfavorable reactions have developed when these small doses have been used continuously for as long as six months," they reported. "So far all adverse effects have been temporary, disappearing on hormone withdrawal or lowering of the dosage."

However, they added: "Only time and further experience will determine the full therapeutic possibilities of cortisone for rheumatoid arthritis. Explorations of its potentialities as a treatment agent are greatly influenced by one fact: The hormone suppresses rheumatic activity but does not cure the underlying disease. Thus, it appears that if antirheumatic effects are to be sustained, cortisone must be given continuously.

"The question as to whether the hormone can be administered safely and effectively for extended periods of many months or years will not be answered positively until there has accumulated greater clinical experience in relation to dosage and methods of administration, greater knowledge regarding its physiologic activities and more information as to the consequences of its prolonged or repeated use."

XUM

CLINICAL PATHOLOGICAL CONFERENCE

From the Case Records of the Medical College of Virginia and the University of Virginia Hospitals

HARRY WALKER, M.D., *Editor*
WILLIAM KAY, M.D., *Associate Editor*

CASE I—PROTOCOL FROM MEDICAL COLLEGE OF VIRGINIA*

Clinical Discussion—J. EDWIN WOOD, JR., M.D.
Presentation of Pathology—G. R. HENNIGAR, M.D.
DR. WOOD:

This patient, a nineteen year old white boy, begins a long illness following a severe automobile injury causing considerable contusion to the head, chest, and abdomen. On the 25th day of this first admission, and following his accident, abdominal swelling, presumably ascites occurred, and responded to rest and diuretic measures. Three weeks later ascites again occurred.

Beginning with this episode his troubles gradually multiplied, culminating in increasing ascites and pleural effusion over the last year of his life, the interval between the original ascites and the final uncontrollable ascites and pleural effusions amounting to about ten years.

Numerous side episodes starting with renal trauma or lower nephron nephrosis and ranging through several pulmonary episodes do not add to or detract from the main issue of recurring ascites and pleural effusion. These and related events catalogue the condition as a circulatory situation.

The distressing episode of mental aberration described in the sixth admission is attributable to lithium administered in the form of a salt substitute which we all used at one time. The toxic properties of this element have been well described elsewhere[1,2] and do not bear directly on the diagnostic problem here.

Were it not for one outstanding feature, the diagnosis of constrictive pericarditis following trauma would at once appeal to all. This has been documented a number of times following non-penetrating chest injury and Warburg[3,4] has noted it on six occasions. The unusual feature here is the large, rather than the usual small heart associated with constrictive pericarditis.

Aside from this discrepancy, recurring ascites and pleural effusion stand as strong indicators of constrictive pericarditis. Other support is found in the repeated improvement from diuretics followed by recurrence of ascites. Also the diminishing total serum protein, particularly diminishing albumin fraction, and finally the electrocardiogram of auricular fibrillation beginning with QRS complexes of reasonably good amplitude progressing to small slurred low voltage complexes. The arrhythmia and the voltage change represent common accompaniments of constrictive pericarditis.[5]

One must, however, consider the other horn of the dilemma, namely the enlargement of the heart and the various murmurs described. The former is important, the latter means little, as systolic murmurs which come and go have little significance and as the one systolic and diastolic combination reported in this case was only temporarily present and could easily have been a pericardial rub. Now as to cardiac size, a consideration of chronic mediastino-pericarditis associated with severe carditis and hypertrophy would be reasonably tenable. Trauma is known to activate rheumatic carditis and this boy had scarlet fever previously. Furthermore, this would account for the systolic and diastolic murmurs at the apex which could be interpreted as murmurs of relative mitral stenosis associated with enormous hypertrophy. This fits less well with the recurring ascites and pleural effusion and the generally slow clinical progression. Ordinarily, activated rheumatic carditis would conclude itself more promptly following the original accident. The electrocardiogram might well be different possibly showing left ventricular strain.

One third consideration can be discussed briefly, namely the traumatic injury to the myocardium equivalent to infarction as described by Bright and Beck[6] later associated with cardiac dilatation and failure. Cases of this type have a relatively brief outlook, and rarely present the ascites and pleural effusion syndrome.

How can the differentation be made then between

*Discussion of cases preserted before the Medical Society of Virginia at Roanoke, October 9, 1950, protocols of which appeared in the October MONTHLY.

chronic constrictive pericarditis following the acute pericarditis of trauma as against chronic mediastino-pericarditis of rheumatic orgin originally activated by trauma? Considerable help would be derived from venous pressure readings during the early years. Continued high venous pressures after relief of congestive failure would resolve the problem at once in favor of constrictive pericarditis. Calcification of the pericardium by X-ray would help also, as would fluoroscopic study. These observations are not available. The question of tuberculosis as a cause of the constrictive pericarditis comes up owing to the several febrile episodes associated with lung and pleural findings. Again no true evidence exists for tuberculosis although it is occasionally activated by trauma and does cause constrictive pericarditis.

Finally, a small heart shadow is not an absolute prerequisite[7] for a diagnosis of constrictive pericarditis and the two layers of the pericardium need not be completely adherent in constrictive pericarditis. It is stated that either the epicardium or the parietal pericardium[8] may be thickened. It would seem reasonable therefore to consider this as an example of constrictive pericarditis (concretio cardis) with thickening of the epicardium for example and pericardial effusion on the outside of this. Failing this explanation of the enlarged heart shadow, constrictive pericarditis with cardiac enlargement is still a reasonable assumption.

If this explanation be correct the usual congestive phenomena will appear in the other organs particularly in the liver which will be characterized by the pseudocirrhosis of Pick.

As it stands absolute differentation between the constrictive pericarditis and chronic mediastino-pericarditis is not possible without certain other observations, and as noted venous pressure readings, the question of pulsus paradoxus and diagnostic pericardial tap for pericardial effusion would be helpful. Clinical observations, however, favor the Pick syndrome.

Passing comment should be made on the terminal event which almost certainly will prove to be a pulmonary embolus from a thrombophlebitis in the left leg.

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ANATOMICAL DIAGNOSIS

Fibrosis and hypertrophy of left ventricle of heart (Chronic Isolated Myocarditis). Dilatation and hypertrophy of left atrium with mural thrombus.

Mixed cardiac and portal cirrhosis.

Anasarca, hydrothorax and peripheral edema.

Infarcts spleen and kidney.

Organizing bronchopneumonia, right upper lobe.

PATHOLOGICAL DISCUSSION (DR. HENNIGAR)

First of all, I would like to say I enjoyed Dr. Wood's discussion of this very elusive type of case. Elusive, both clinically and pathologically speaking.

At autopsy, the heart (see Figs. 1 and 2) was

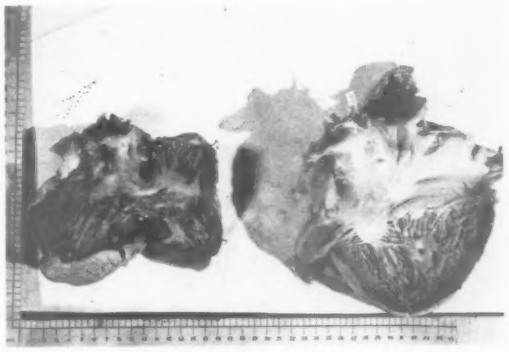

Fig. 1 The heart compared with a normal organ. Note the huge atrium.

enormous, weighing 650 grams. The increase in size was due to hypertrophy of the left ventricular myocardium, as well as to the huge, dilated left atrium. The pericardium was smooth and shiny. The myocardium of the left ventricle measured 2.3 cms. Throughout there was a very fine scarring extending from base to apex. The left atrium was immensely dilated and hypertrophied. A mural thrombus 2 cms. in widest diameter was present in the wall. The

Fig. II The left wall of the ventricle is hypertrophied. The coronary orifices are patent.

right ventricle showed slight hypertrophy.

Careful inspection of the endocardium and valves revealed no special change.

The liver weighed 2,330 grams. On cut section, the capsule was thickened and wrinkled with small 2 to 5 mm. nodules on the surface. On cut section, the nodules were pale in color and connective tissue trabeculae separated them.

The spleen weighed 260 grams. The surface inspection was not remarkable on cut section, an old pale infarct 3 cms. in longest diameter was seen.

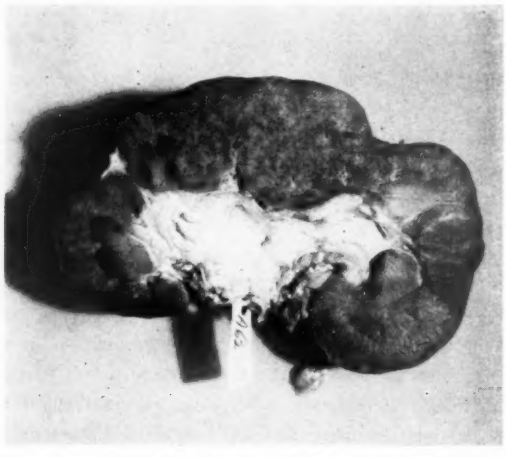

Fig. III The cortical markings are indistinct and there is a pale mottling of the whole parenchyma. The pelvis and calyces are wrinkled and opaque.

The right and left kidneys weighed 240 and 290 grams respectively. The capsules stripped readily displaying deeply grooved, bizarre, sizable scars. On cut section (see Figs. 3 and 4) the cortico-

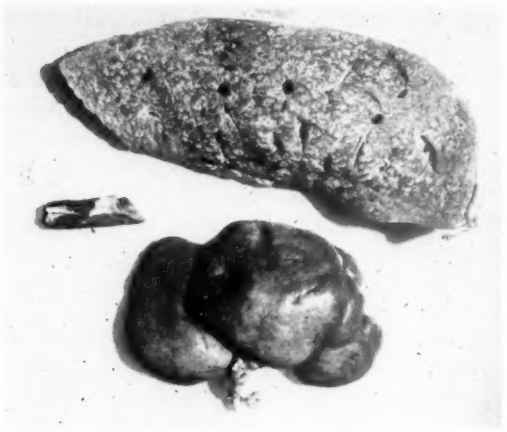

Fig. IV Small yellow nodules are scattered throughout the liver, there is also fibrosis present. The intrahepatic veins are dilated and thick walled. The spleen shows an old infarct. Deep pitted scars are seen on the surface of the liver and fetal lobulation has persisted.

medullary junctions were not distinct and the pelves were granular and thickened.

The remainder of the gross examination revealed no special pathological changes. Permission to examine the brain, spinal cord and eyes was not granted.

Microscopically, the myocardium was the "seat" of diffuse fibrosis accompanied by marked hypertrophy and vacuolation of myofibrils. Numerous sections were taken from various regions of the left myocardium and all showed this same change. Sections of the right ventricle and left atrium revealed slight hypertrophy and minimal scarring. The vessels appeared normal.

Microscopic examination of the liver (see Fig. 5) revealed small nodules of liver cells separated by connective tissue. Many of the cells contained fat vacuoles. Angiogenesis and lymphocytic infiltration were prominent in the portal areas.

Microscopic examination of the spleen and kidneys reveal small infarctions. In addition, the transitional epithelium of the pelves and calyces were hyperplastic and the tubules lined by tall epithelium with granular cytoplasm. Many of the glomeruli were obliterated by scarring and lymphocytes in the interstitial tissue prominent.

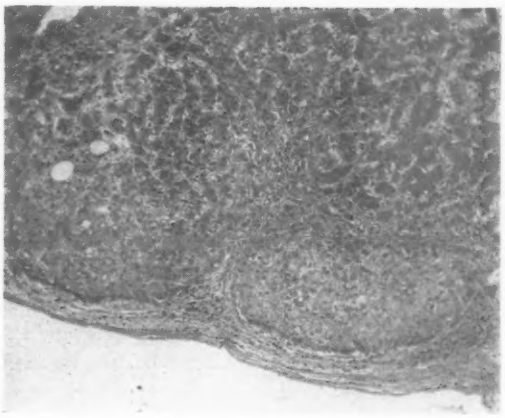

Fig V Liver. Two demarcated nodules are seen under a thickened capsule.

The lungs microscopically revealed the signs of long standing broncho-pneumonia and interstitial fibrosis (see Fig. 6).

CAUSES OF DIFFUSE MYOCARDIAL FIBROSIS

1. Coronary insufficiency with or without thrombosis and infarction.
2. Rheumatic carditis.
3. Congenital Heart Disease.
4. Severe Anemia.
5. Post Diphtheria myocarditis.
6. Syphilis.
7. Tuberculosis.
8. Subacute bacterial endocarditis.
9. Traumatic myocarditis.
10. Arsenic.
11. Sulfa.
12. Poliomyelitis, Scrub typhus and a variety of mycotic diseases.
13. Lupus erythematosus.
14. Fiedler's (isolated) myocarditis. Diffuse or granulomatous. Acute, chronic or healed.

Neither grossly nor microscopically (see Fig. 7)

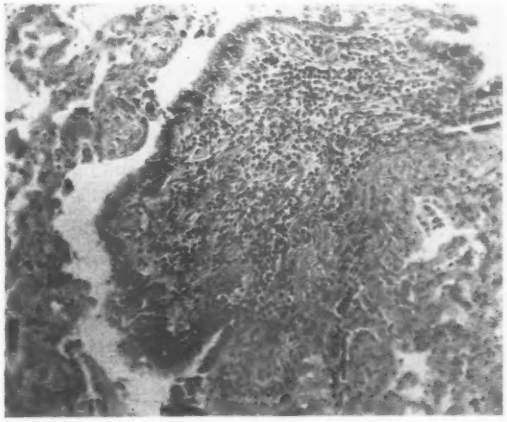

Fig. VI The bronchial wall is infiltrated with lymphocytes and fibrosis is prominent.

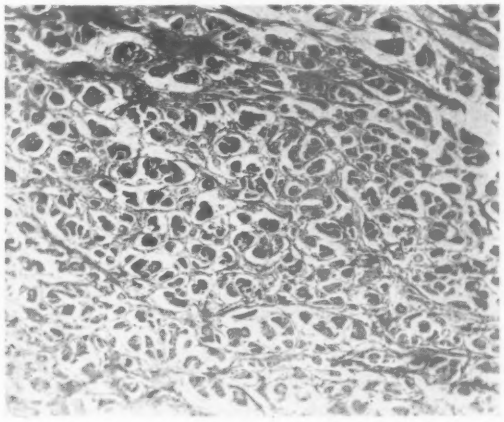

Fig. VII-A A diffuse fibrosis is present.

This case is one of idiopathic chronic myocarditis or interstitial fibrosis. Since Fiedler's[1] publication of a group of cases characterized by diffuse myocarditis of unknown etiology a great interest has been shown in these cases. In his group, the interstitial or supporting tissues of the myocardium contained polymorphonuclear leucocytes, lymphocytes, histiocytes and giant cells. This case today shows none of the acuteness of that type of lesion and we thus propose it as a chronic or healed Fiedler's myocarditis. It is obvious that this type of heart disease is simply one where we have not been able to discover the etiological agent.

is there anything specific or pathognomonic for this type of myocarditis.

We have seen identical pictures in lupus erythematosus, congenital syphilis and post diphtheria myocarditis.

We believe this case to be identical with those described by Levy and Rousselot[2]. The cause of the sudden death was probably attributable to ventricular fibrillation as the result of the extensive fibrosis. The majority of cases of these kind die a sudden death.

The sequence of events in this case may be explained as shown in Fig. 8.

XUM

Fig. VII-B Note the large bizarre nuclei so often observed in marked hypertrophy of the myocardium.

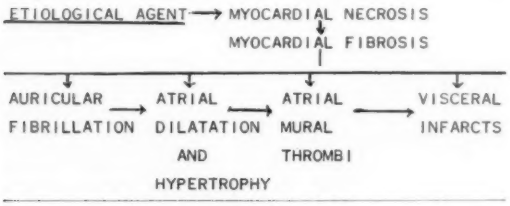

Fig. 8

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CASE II—PROTOCOL FROM UNIVERSITY OF VIRGINIA

Clinical Discussion—KINLOCH NELSON, M.D.,
Presentation of Pathology—J. F. A. MCMANUS, M.D.

DR. NELSON:

I appreciate greatly the opportunity to appear here and to study the very interesting record supplied from Charlottesville. Certainly no patient has ever been more thoroughly studied nor have such studies been so generally negative. As we read through the data we note observations which make me feel that the discussion should hinge around the kidneys which are certainly not functioning properly. Thus the basic problem is: do we have intrinsic kidney disease, do we have disease elsewhere in which kidney function is impaired secondarily, or do we have both? From this viewpoint I will now review the protocol.

The history shows a 71 year old female with vague weakness for six months or so, and more definite weakness, nausea, anorexia, occasional vomiting, and *marked* weight loss over a five-week period. We note a change in bowel habit more or less coincident with the onset of her illness. All these symptoms suggest malignant disease of the gastrointestinal tract, but this possibility can be discarded in view of the repeatedly negative x-rays, the absence of significant anemia, the negative proctoscopic examination, the absence of achlorhydria, and the stool examinations which were, for the most part, negative for blood. The "batting average" of these studies in ruling such cancer in or out is so high that we must omit it from further consideration. I expect that the history alone would have led to suspicions of psychoneurosis; without knowledge of the background, home situation, personality, etc., we can say little more along this line, except that such a marked weight loss is against this possibillity as are subsequent events, of course.

The physical examination was not helpful. The blood pressure of 150/84 is not exciting. The enlarged thyroid, kyphosis, and pelvic findings have little significance. The cardiac murmur is worrying because I cannot too well fit it and its subsequent course into my reasoning. Surely we should not be much concerned about a systolic murmur in an elderly woman with slightly elevated blood pressure. However, such murmurs are seldom recorded as "loud" or "to the left of the sternum", and it may well be that this finding is of great importance. I will refer to it again.

In the laboratory the blood counts are not helpful; they may be regarded as normal or evidence of slight dehydration. It makes me a little uneasy that no differential count is given but I presume this was an omission in the hospital (later rectified) as I was assured by Dr. Wood that he (I quote) "included all data available from the chart and none was withheld". The urine was scarcely abnormal and certainly not consistently so; a concentration to 1.016, 2+ albumin in one of three specimens, a few casts in one specimen, and a normal catheterized specimen are hardly compatible with any known intrinsic kidney lesion. However, there must be disturbed kidney function as manifested by nitrogen

retention and depressed phthalein excretion. These kidney problems will be discussed below. The blood proteins and sedimentation rate were normal and the blood sugar of doubtful meaning; it is noted that the first blood sugar of 100 might represent a low figure for this patient as a later one was 154; that is, as she improved the blood sugar returned to her normal level, possible evidence that some disease was upsetting her sugar metabolism. I feel that this is grasping at straws but one cannot make bricks without straw. The gastric analysis was of little moment. The agglutination tests were negative. I doubt if it would have occurred to me to get these done except that she ran a fever from 99-100 and had a good many vague symptoms. As you know, a rumor has gone around for some years that apparent neurasthenics with low-grade fever may have chronic brucellosis. Such a condition must be rare as we have tested many similar patients in the out-patient department with negative results. In any case I do not see how chronic brucellosis could have been responsible for this lady's illness and we will discard it. The electrocardiogram was not considered abnormal and the x-rays have been mentioned already.

Her course in the hospital is not informative. The low-grade fever suggests nothing much. Her depression may be quite significant. Her weight remained stationary, which may mean that the history of weight loss was an error (unlikely I think), or that she was improved by therapy (type unknown), or that her disease abated.

It would be interesting to know what diagnosis was recorded at this time.

Her progress at home brings out nothing striking. The apathy is noted again. Also it is worthy of mention that her second admission was precipitated by a belching attack. It may well be important that her pulse was rapid though her fever was still relatively insignificant. The change in the heart murmur is quite alarming to me because I cannot see its meaning. We might mention bacterial endocarditis at this point. Surely we must think of this in a patient with low-grade fever, a changing heart murmur, and disturbed kidney function, but I cannot believe it could be present in the absence of a palpable spleen, blood in the urine, petechiae, and organisms in many blood cultures; also the physical examination and x-rays disclose no enlargement or unusual contour of the heart shadow; nor is the course of the illness suggestive of this disease. I realize that all too often the bacteria cannot be isolated from the blood and that almost any finding or group of findings may be absent, but I will exclude it, nevertheless. I cannot then explain this changing murmur unless it reflects somehow the circulatory upset necessarily accompanying the electrolyte disturbance.

The physical examination was little changed except for a decline in the blood pressure, a very significant observation, we believe. It would be nice if we knew the subsequent course of the blood pressure but this is not available. It is again noted that the heart size was within normal limits by physical examination and x-ray. If we pursue the reasonable assumption that the blood pressure had been moderately elevated for some years, we might expect a heart shadow suggesting enlargement or at least rounding off of the left ventricle. Thus the presence of a heart of normal size suggests that it is unexpectedly small. Again we grasp at straws.

In the laboratory work on the second admission I will first take up those studies which seem of little significance. The stools may be disregarded. The blood counts are not helpful though the shift to the left might make one think of inflammation, as does a finding of 13,000 in one of two white blood counts. The bone marrow was normal. I wonder why this was done. Whenever I see a study, the reasons for which are not clear, I try to put myself in the place of the bedside clinician because his actual observation will often lead him to examinations not readily foreseen when one considers only the cold data. By so doing I have occasionally thought of possibilities I had not previously considered. No such light is shed here. The efforts to find some infectious agent by blood cultures, urine cultures, and agglutination tests were fruitless. I do not believe any ordinary germ could have escaped the clutches of the laboratory and I will exclude infectious disease with some reservations. It is not necessary to detail certain factors which, at first glance, suggest Weil's disease. When we recall that this disease has been reported, especially in Europe, in the absence of jaundice, we might be uneasy about the omission of any specific study along this line. However, the course of the illness does not lead me to consider it seriously. This brings us to the tuberculin test. A somewhat brief experience with this type of exercise has

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made me realize that we should never come to any conclusion without seriously considering tuberculosis, cancer, and lymphoma. For these last two there is no shred of evidence and I will discard them at once; tuberculosis, however, cannot be so lightly dismissed. The negative tuberculin test may be regarded as evidence of the absence of tuberculous infection, now or ever, or, quite the contrary, it occurs in relatively acute overwhelming disease. The possibility of generalized tuberculosis here is somewhat attractive, as we have recently seen a similar case in a man of 70. Also we note the prominent hilar shadows and "non-specific" pulmonary fibrosis. However, I do not believe this patient has generalized tuberculosis, chiefly because of the kidney status. The electrocardiogram and x-rays are within normal limits. We may assume that no calcification was noted in the kidney or adrenal areas. The spinal fluid and skull films were normal. I wonder why the latter were done; perhaps brain tumor was suspected. The serological tests for syphilis were negative. The liver function tests were normal, as was the blood sugar (note that again it is lower than the value recorded when she improved before). The blood proteins were somewhat reduced. The pancreatic function tests are difficult to evaluate. The lipase was normal. The amylase was high one day and normal the next. I cannot see the significance of this and am forced to exclude intrinsic disease of the liver and pancreas.

The gastric analysis is not important except that a patient who has free acid and is vomiting a great deal loses large amounts of chlorides, thus reducing the blood chlorides and raising the CO_2, both prominent findings in this case. However, we do not get the idea that vomiting was profuse, frequent, or regular though such may have occurred in her latter days.

We come now to the kidney, where, as stated originally our problem seems to center. Observe that no where in the record is there any mention of urinary output. We have no knowledge of its increase or decrease, possibly very important points. We recall that in the first admission the urinalyses were variable and of doubtful significance, that the PSP was 35, and the blood urea 57, later dropping to 29, perhaps as the result of hydration and other treatment. In the second admission we again find slight and inconstant variations from normal urine, observations which to me eliminate intrinsic kidney disease of any type, with one exception, namely salt losing nephritis.

Thorn[1] described two cases (I can find no others) considered to be in Addisonian crisis because of their shock-like state and characteristic electrolyte pattern. Their urinalyses were normal. Both responded to massive doses of sodium chloride, and later bicarbonate, whereas specific treatment of adrenal cortical insufficiency was ineffectual. Both later died with chronic kidney failure. The data here do not suggest this course.

The blood urea was elevated and variable (perhaps related to treatment) with marked terminal rise; the urea clearance was 38.6% of normal; and the phthalein excretion was low, particularly that in fifteen and thirty minutes. These findings are inescapable evidence of depressed kidney function yet we believe the kidneys were anatomically normal. We must then seek for disease elsewhere which affects these kidney functions. Such conditions must also have a persistently low blood chloride (unresponsive to treatment, type unknown) and a somewhat high CO_2, bearing in mind that these latter findings may be related to the vomiting of hydrochloric acid, and the failure to take in salt because of nausea and anorexia. It is generally believed that the blood sodium level can be estimated reasonably accurately by adding the chloride level (77) to the CO_2 (37) plus an arbitrary figure of 10. By so doing we get a value of 124, indicating that the blood sodium is also quite low. We would very much like to know the blood potassium level but it is not at hand.

Thus we narrow our search to extra renal conditions where kidney function is depressed, nitrogen is retained, and blood sodium and chlorides are low. Referring to Cantarow and Trumper[2] we may list these possibilities: Loss of gastro-intestinal secretions; shock; burns; intestinal and pyloric obstruction; infectious diseases; severe liver disease; diabetes mellitus; excessive sweating; adrenal cortical insufficiency; and hyperparathyroidism.

At a glance we may reasonably eliminate most of these. However, we are still left with infectious disease and adrenal cortical insufficiency. The latter is especially attractive but for one almost insurmountable obstacle, namely the absence of pigmenta-

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tion. I have not personally recognized a case in which it was absent; some authors state that it is always present. However, proven cases are recorded without it.

In following the course of the case we find that she went progressively downhill with anorexia, nausea, vomiting, marked apathy, rising temperature, and low blood chlorides. Treatment was unsuccessful in correcting the hypochloremia. It would be interesting to know what this treatment was. Was it intravenous fluids, adrenal cortical extract, or desoxycorticosterone, or some combination of these? Also since she had fever she must have gotten antibiotics.

To sum up, in our final conclusions we must review four possibilities.

I. Infection, such as generalized tuberculosis, as suggested by weakness, weight loss, nausea, vomiting, rising fever, dehydration, and low blood chlorides. Many studies failed to yield any clue as to etiology or location.

II. Chronic nephritis, as manifested by the same symptoms, the abnormal kidney function tests, the nitrogen retention, the hypochloremia, and inferred hyponatremia. Against this diagnosis are the many more or less normal urines, the apparently declining blood pressure, the normal intravenous pyelograms, and the course of the disease. We cannot absolutely exclude salt losing nephritis but, judging from the only available reports, this seems a very remote possibility.

III. Adrenal cortical insufficiency. There is no suggestion of other gland disturbance particularly, so that we do not believe primary pituitary failure should be considered. Primary adrenal failure, or Addison's disease, is strongly suggested by the weakness, apathy, gastro-intestinal symptoms, precipitation of hospitalization by a belching attack, marked weight loss, declining blood pressure, slight electrocardiographic changes, heart of normal size, suggestion of disturbed sugar metabolism, nitrogen retention, abnormal kidney function tests, low blood chlorides, and inferred low blood sodium. Further the course of the case was rather rapidly downhill and the terminal rise in temperature has often been noted in this disease without evidence of infection at autopsy. The absence of pigmentation, the low-grade fever throughout, and the changing heart murmur are very annoying, especially the lack of pigmentation. However, had such changes been present we would not be discussing this case. If we assume Addison's disease we must consider either tuberculosis or atrophy of the glands. Favoring the former are the low-grade fever, and statistics; suggesting the latter are the absence of evidence of tuberculosis elsewhere and the negative tuberculin test.

IV. There is, of course, the possibility of some combination of factors, such as both renal and adrenal disease; terminal infection as in the lungs cannot be entirely excluded.

Having put off the evil hour as long as possible I will conclude by making a diagnosis of Addison's disease due to atrophy of the adrenal glands.

REFERENCES

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2. Cantarow and Trumper: Clinical Biochemistry, Fourth Edition, W. B. Saunders Company, pages 103-105 and 231-239.

PATHOLOGICAL DIAGNOSIS

Adenocarcinoma, tail and body of pancreas with local extension and metastases to liver, kidney, peritoneum and lymph nodes.

Confluent lobular pneumonia, right lower lobe.

Generalized arteriosclerosis, moderate.

Cardiac hypertrophy with scarring.

Chronic cystitis.

Bilateral chronic pyelonephritis, moderate.

PRESENTATION OF PATHOLOGY—DR. MCMANUS

The body was that of a well developed, elderly, white female showing a 4 x 3 cm. decubitus ulcer on the posterior thorax.

Viscera revealed an adenocarcinoma arising in the body and tail of the pancreas, with local extension to the left kidney, stomach and surrounding peritoneum, especially the transverse colon. Also noted were metastases to the liver and regional lymph nodes.

The kidneys showed changes consistent with the findings of chronic pyelonephritis, and the bladder showed chronic cystitis.

There was moderate arteriosclerosis and hypertrophy of left ventricle of heart with scarring.

Confluent lobular pneumonia was seen in the right lower lobe of lung.

The adrenals showed no significant change, though it is quite conceivable that altered function might exist without demonstrable changes.

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PUBLIC HEALTH

L. J. ROPER, M.D.
State Health Commissioner of Virginia.

The report of the Bureau of Communicable Disease Control of the State Department of Health for September 1950 as compared with the same month in 1949, and for the period of January through September 1950 as compared with the same period in 1949, is as follows:

	Sept. 1950	Sept. 1949	Jan.-Sept. 1950	Jan.-Sept. 1949
Typhoid and paratyphoid	11	14	68	104
Diarrhea and dysentery	540	983	3,250	4,901
Measles	65	39	2,541	21,276
Scarlet fever	55	58	674	514
Diphtheria	30	14	132	106
Poliomyelitis	331	84	878	247
Meningitis	1	7	94	73
Brucellosis	9	13	49	56
Rocky Mountain spotted fever	7	11	65	97
Tularemia	8	3	33	28

IMMUNIZATION IN COMMUNICABLE DISEASE CONTROL

With the opening of schools and the coming of the fall and winter season when we anticipate a rise in the incidence of the communicable diseases of infancy and childhood, it is well for physicians and public health workers to take inventory of the degree of community protection against such diseases.

The accompanying table shows the decline over twenty years in three communicable diseases for which specific immunizing agents are available:

The decline in incidence as shown by the above list is mainly due to the continuing immunization activities of practicing physicians and public health authorities, but it is essential to realize that the job is not complete as long as new crops of non-immunes are coming on and the ones previously immunized have lost some of their early acquired protection. Immunization of the young, with later booster doses to maintain immunity at high levels, is the basis of effective control. In this continuing control "Eternal vigilance is the price of safety".

In an immunization clinic held in one of the Virginia Counties it was startling to find how many school principals and teachers had never been vaccinated against smallpox. The principal of one school, who also drives the school bus, had such a marked reaction when vaccinated that he could not drive the bus and a holiday had to be declared for three days. The pupils were unanimous for compulsory vaccination, even though late.

The public is inclined to regard whooping cough, along with chickenpox and mumps, as one of the minor diseases of childhood. A look at the record shows that in 1948 there were 41 deaths from whooping cough as compared to 32 from poliomyelitis. The importance of early immunization with whooping cough vaccine should be stressed, since children under two years of age are most susceptible to fatal attacks. Immunization should be begun by the second month and certainly not later than the sixth.

INCIDENCE OF CERTAIN COMMUNICABLE DISEASES IN VIRGINIA—1930-1939

Year	1930	1931	1932	1933	1934	1935	1936	1937	1938	1939
Diphtheria	2,210	4,866	2,057	2,139	1,989	1,502	1,252	1,294	1,794	1,474
Smallpox	264	124	17	10	13	10	4	6	5	0
Whooping Cough	8,389	6,987	11,777	2,607	5,449	3,355	1,924	4,324	3,803	3,054

1940-1949

Year	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949
Diphtheria	709	754	699	395	329	577	548	273	265	220
Smallpox	5	0	0	0	1	0	0	0	0	0
Whooping Cough	2,902	4,327	2,405	5,060	2,719	2,749	3,058	4,234	2,271	1,378

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Medical Society of Virginia Cancer Committee

Chairman, GEORGE COOPER, JR., M. D.
Medical School Building, University, Virginia

Reprints of this and preceeding Bulletins may be obtained from this office
November 1, 1950

Case Reports

A 48 year old farmer began losing strength and energy. In a period of about three months, he became too weak to work. Simultaneously, numerous occipital, cervical, axillary, inguinal, and femoral nodes grew firm enough to palpate, though none were over 1.5 cm. in diameter. For the next three years, this man's condition remained about the same. At times he felt a little stronger than at others, but he was never able to resume normal activities. His condition worried him and he became depressed and nervous. During the three years, he sought the assistance of three doctors. The first two included no blood studies in their work-up, told him his troubles were due to a nervous condition, and prescribed sedatives. The third doctor checked his hemoglobin, told him he was anemic as well as nervous, and prescribed a "blood tonic" in addition to the sedatives.

Finally the patient developed a violent pain in the right shoulder girdle, extending down the arm into the hand. Hospitalized under the care of a cardiologist, a full work-up was done at last. Pertinent findings included a normal electrocardiogram, generalized peripheral lymphadenopathy, moderate enlargement of the spleen and liver, enlargement of the hilar nodes demonstrated by an x-ray examination of the chest, and a white blood count of 55,000 with 80 per cent lymphocytes. The only detail not included in standard textbook descriptions of chronic lymphatic leukemia was the right shoulder pain radiating down the arm. This symptom was presumed to be due to lymphatic infiltration of the brachial plexus, a presumption which was proven by the prompt disappearance of the pain when roentgen therapy was delivered to the shoulder girdle. The axillae, mediastinum, and spleen were irradiated also. Within two weeks, the patient was asymptomatic, the size of the lymph glands and spleen was sharply reduced, the blood picture was approaching normal, the depression was greatly relieved, and strength was returning. One month later, the occipital nodes suddenly enlarged to an average diameter of 3 cm., to the accompaniment of severe headaches. X-ray therapy again afforded excellent relief. The patient has been seen periodically since. For six months, he has been in good condition and has been able to manage his farm again.

Comment: 1. A hemoglobin determination, a red blood cell count, and a white blood cell count are an essential part of a routine physical examination.

2. A diagnosis of psychoneurosis should never be made before the possibility of organic disease has been ruled out by complete, careful investigation of a patient's condition.

3. The above case report illustrates once again the monumental oversights physicians will inevitably commit when they begin to make a habit of taking "short cuts." For this unfortunate patient, the "short cuts" meant that three years of his few remaining ones were spent in unnecessary mental and physical distress.

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MENTAL HEALTH

Joseph E. Barrett, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals.

Convulsive Therapy and Related Psycho-Physiological Phenomena*

The two types of convulsive therapy most widely employed in Virginia mental institutions are electrocoma and deep insulin coma. Metrazol shock has almost been abandoned. The term "shock" is unfortunate in that it frightens practically all laymen and even most physicians.

Electro-convulsive therapy is administered with a variety of instruments and techniques. Its several forms are also referred to as electro-shock and electro-stimulation. Electronarcosis is a modification of one of these with considerably more restricted usage. These therapies are employed in all major psychoses (organic and functional), psycho-neuroses, personality disorders, alcoholism (not in states of intoxication), drug addictions, and barbiturate intoxication (for stimulation of respiratory centers). They are generally used before deep insulin shock is resorted to and only discarded in favor of the latter when it becomes apparent that desirable results will not ensue.

Deep insulin shock is a much more drastic therapy than the electrocoma therapies. It is reserved usually for schizophrenics between the ages of 18 and 40 who are in good physical condition and who have been shown to be refractory to electrocoma.

Both electrocoma and insulin shock are employed with widely varying results in post-lobotomy syndromes. Electrocoma is used with moderately good results in such psychosomatic disorders as spastic colon, asthma, pylorospasm, functional vomiting, tension-type headaches, etc. Even migraine, diabetes mellitus, and thyrotoxicosis appear to improve with this line of therapy.

Convulsion brought about by electrostimulation has essentially the same physiological modus operandi as epilepsy. The electrochemical impulse which is set up by the stimulation spreads in concentric and ever-widening waves in all directions from the area of stimulation until the hypothalamic cortex is reached. From this control center of the sympathetic system, a chain of stimulatory electrochemical impulses is set into action and passed down the sympathetic chain setting all the sympathetic functions of the organism into frenzied activity for a short time. This process does not exclude the sympathetic innervation of the adrenal cortex which immediately sets about to produce ACTH in the pituitary in large quantity. One electrocoma treatment is said to produce many times more than the average dose of 25 milligrams of parenterally administered ACTH. This is likely a large part of the explanation of the improvement in certain types of arthritis seen as the result of electrocoma.

Despite all the theoretical, as well as practical, implications of electrocoma, they must not be magnified to the exclusion of psychological factors involved in the treatment situation and of other physiological phenomena incidental to the procedure. Among the latter may be mentioned the postulated transient physiological separation of the frontal cortex (with its ideational centers) from the thalamus (with its affective centers). This is said to be brought about by a transient blocking oedema of the fibers of the fronto-thalamic radiation producing temporarily that which is produced more permanently by lobotomy. This postulation is open to some question in the opinion of the writer, but it must certainly be admitted that some very beneficial dissociation of affect is seen as the result of electrocoma and insulin shock. Certainly, we must also consider the role of the metabolite, lactic acid, which is elaborated in the muscles in large quantities in convulsive therapy resulting in the production of relaxation and in the diminution of tension, anxiety, and depression. Duke University investigators have advocated and used extensively their lactic acid-sodium lactate therapy in the depressions. The effects of altered oxygen-carbon dioxide ratios should receive attention as well as of the transient cortical telangiectasis both of which occur during electrocoma, insulin shock, and gasconvulsive therapy. There are also a good many other psycho-physiological phenomena in connection with electro-coma which cannot be discussed here due to lack of space.

*Article prepared by C. Earle Johnson, Jr., M.D., Superintendent, DeJarnette State Sanatorium, Staunton, Virginia.

It would seem that great strides are now in the making in the dove-tailing of physiologic with psychiatric science. As recently as five years ago, leading psychiatrists and internists of the country were unable to comprehend or to endorse the etiological approach of totality so strongly advocated by the school of psychobiology and its founder, Adolf Meyer. Now, it would appear that the purely organic and purely functional schools are fading into the past.

Of the psychological factors entering into the electro-coma treatment situation, a great deal has been written, especially by the psychoanalysts. These concepts, while they may not be considered in detail here, are undoubtedly of great importance. The successful and best administration of electrocoma and of deep insulin shock involves a great deal more than the simple mechanics of the procedures.

If the writer has indicated undue enthusiasm for convulsive therapies, it is hoped that extenuating circumstances may be found in his personal experience of somewhat more than 10,000 such treatments. It is emphasized that despite such experiences by different psychiatrists, the surface has been barely scratched in the ramifications, implications, and potentialities of the various types of convulsive therapies.

BOOK ANNOUNCEMENTS

Friend of the People. The life of Dr. Peter Fayssoux of Charleston, South Carolina. By CHALMERS G. DAVIDSON. Published by the Medical Association of South Carolina, Columbia, 1950. vii, 151 pp. price $2.75.

This attractive little book is very timely. Little is known about this Revolutionary hero of South Carolina, and what has been published about him is inaccurate. The *Cyclopedia of American Medical Biography*, for instance, gives him a middle name and a birthplace in Southern France and does not mention his first wife.

The author, Dr. Chalmers G. Davidson, is a professional historian and his story is well documented. Much of his information is obtained from Fayssoux's correspondence with Benjamin Rush. The two physicians were schoolmates in Edinburgh and carried on a correspondence until Fayssoux died of apoplexy, February 1, 1795. Two of Fayssoux's daughters apparently had Hansen's disease. The father's description of his daughters' disease in his letters to Rush and his attempt to get medical help is one of the most interesting parts of the book. The part that Fayssoux played in that critical period of the nation's history before the adoption of the constitution is especially interesting at this time when the constitution is fast losing its position of prime importance in our fundamental law.

M. P. R.

A Century of Medicine in Jacksonville and Duval County. By WEBSTER MERRITT. University of Florida Press. Gainesville. 1949. 201 pp. Price $3.50.

There is need of state and county medical histories for two reasons. One is as a reference work where one can find biographical information about doctors who are not quite important enough to be listed in the usual biographical dictionaries. The other is that it may be an interesting story of a too-frequently neglected subject. Very often the first object overshadows the second. Dr. Merritt has happily combined the two and his book makes good reading even for those who have no local interest. The present work is important because in the Jacksonville fire of 1901 many records were destroyed and much important information would have been lost forever had not someone with historical training undertaken to gather it and critically analyze it at this time.

Particularly interesting are the struggles of Jacksonville with epidemics of scarlet fever, smallpox and yellow fever. The difficulties the local health authorities had with commercial interests and the newspapers strike a familiar cord. How the problem of public water supply was solved is always of interest to thousands of small communities throughout the country. Probably the most interesting chapter is that dealing with the impact of the Civil War on Jacksonville and the surrounding country.

The book is well illustrated and has an attractive format.

M. P. R.

EDITORIAL

William Henry Welch (1850-1934)

ONE hundred years ago a boy was born in Norfolk, Connecticut, who was destined to revolutionize the teaching of medicine throughout the entire world. "Dr. Welch" was no new name in New England. For three generations there had been one to three Doctors Welch in Norfolk. The inhabitants of Lichfield County must have been surprised therefore, when the son of Dr. William Wickham Welch in 1866 went to Yale to study the classics instead of medicine. God must have had other plans for William H. Welch, for when he was unable to find an attractive post in Greek, he reluctantly turned to medicine. He entered the College of Physicians and Surgeons in New York, graduating in 1875. From then on his course was as consistent and purposeful as if he had been following a blue print.

When Dr. Welch graduated in medicine, medical education in this country was anything but inspiring. There was not a laboratory in the country where one could study a problem in physiology, pharmacology, pathology or even microscopic anatomy. There was, of course, no such thing as bacteriology.

In 1876, Dr. Welch went to Europe for postgraduate study. In Strasbourg he found that he could not take v.Recklinghausen's course in pathological histology because he had no knowledge of histology. He had to be content with a course in gross pathology and a course in histology under Waldeyer, and to fill in he took a course in physiological chemistry under Hoppe-Seyler. In the autumn he enrolled in Prof. Wagner's pathological institute in Leipzig, and more or less by accident found his way to Ludwig's laboratory where he worked on a problem of the distribution of the nerves of the heart. It was at Leipzig that he first met John Shaw Billings who even then was gathering information preparatory to starting the hospital and medical school at Johns Hopkins. They had an evening together in Auerbach's Keller, and Billings advised Welch to spend another year in Germany. At Ludwig's recommendation he next went to Breslau and entered Cohnheim's Laboratory. Here he worked out the mechanism of edema of the lungs. He was in Breslau when Koch came there to demonstrate his work on anthrax to Cohnheim, but Welch does not seem to have been impressed. At Cohnheim's suggestion he went to Prague to visit Klebs. Next, he went to Vienna to take some "practical courses" in case he would have to practice medicine for a living when he returned to America. He stayed in Vienna only 6 weeks, and then went back to Strasbourg for the course under v.Recklinghausen in pathological histology that he wanted to do at first. After a sight seeing trip to Paris and London, he sailed for home on February 6, 1878.

The next six years were spent in trying to sell to New York the knowledge he had acquired in Germany. They were hard, discouraging years, but he learned what America needed in the way of scientific medicine and how to organize courses. Above all, he learned to teach. When President Gilman and John Shaw Billings were ready to start their medical school in Baltimore, Dr. Welch was prepared for the job. He was the first professor appointed to the medical faculty and he had a voice in the selection of the other members. He was granted a year's leave to go abroad to buy equipment and to study the new science of bacteriology. He applied to Koch for permission to work in his laboratory, but Koch's laboratory belonged to the Army and it was necessary to get permission of the Minister of War. Koch advised him to study under his pupil Frobenius in Munich. This he did, but of more importance was his contact with Pettenkofer and his public health work, and with v.Ziemssen who had

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the first clinical laboratory in Germany. He was also impressed with the fine laboratory of the veterinary school. Later he studied under Flügge in Göttingen and finally under the master himself when Koch transferred to his new laboratory. On his way home he visited England and spent some of the Hopkins money buying apparatus in Cambridge.

The story of Dr. Welch at Hopkins is well known. His own investigations, particularly in thrombosis and embolism and the discovery of the *Clostridium welchi* were outstanding, but of more importance, he trained pathologists for the nation. He took great interest in community health projects and rendered invaluable service in planning Baltimore's sewerage system. For years he was President of Maryland's State Board of Health. Of even greater importance from the standpoint of medical education was his presidency of the Board of Scientific Directors of the Rockefeller Institute which position he held from 1901 to 1933. He was also chairman of the Executive Committee of the Carnegie Institute of Washington, Chairman of the Advisory Council of the Milbank Memorial Fund, and Chairman of the Advisory Council of the Rockefeller Foundation's Sanitary Commission. The School of Public Health at Hopkins, the first of the kind in the world, was a natural result of Dr. Welch's interests in medical education and in public hygiene. The same may be said of the Institute of Medical History at Johns Hopkins. The important thing, however, was that Dr. Welch's wisdom and sound knowledge of the subjects insured their being established on a firm basis of university teaching. President Bronk, in the recent Welch memorial issue of the Johns Hopkins Hospital Bulletin, aptly summarized the situation by calling William Henry Welch the greatest statesman of the world in the field of public health. He might well have added and of medical education.

Recent Statistics on Draftee Rejections

DRAFTEE rejections for physical reasons in August were about 20 per cent lower than they were in July. Rejections for all reasons were 49.4 per cent in August as contrasted with 69.4 per cent in July. Twenty-two and a half per cent of the total number examined was for physical defects. According to Gen. Hershey the drop in percentage of rejections for physical reasons (29 per cent) was due largely to a change in dental standards.

We have no quarrel with the Army for these changes. It knows, no doubt, the dental requirements of Army chow. What we object to is the use of such statistics for purposes other than for the purpose for which they were gathered. The next thing we will hear is that the country needs more and better medical care as is shown by the great numbers of rejectees on account of physical defects. Had Hollywood Producers formulated the dental standards the percentage of rejections would probably have been 98 per cent. On the other hand, had the standards been adjusted to farm, factory or counting house requirements, the percentage would have been a great deal lower, possibly 15 per cent. Yet these Army figures will be used before various committees of the Congress without any reference to the varying standards upon which the examinations were based.

It is interesting that there was little change in the percentage of rejections for mental and educational reasons—21.9 per cent in August and 20 per cent in July. Gen. Hershey has been urging an easing of physical and mental-educational standards by the Armed Forces, pointing out that in World War II the total rejection rate was between 34 and 35 per cent, as contrasted with the August 1950 rate of 49.4 per cent. In the same paragraph, he is reported to have said that less than one-third of the men deferred to take pre-medical training ever enter the medical school. Does this mean that they are not up to the present physical and mental-educational standards?

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Our New President

CHARLES LYDON HARRELL, M.D.

AT the Annual Session of 1950, in Roanoke, Charles Lydon Harrell was installed as President of the Medical Society of Virginia.

Doctor Harrell was born March 19, 1883, in Gates County, North Carolina. He attended Randolph-Macon Academy and Randolph-Macon College in Virginia, later receiving his medical degree in Richmond in 1909, at the University College of Medicine, which afterward affiliated with the Medical College of Virginia. After hospital experience at the Virginia Hospital, in Richmond, and the New York Post-Graduate Hospital, he began the practice of medicine in Norfolk in 1910. In 1911, he was married to Miss Ethel Toone, of Richmond, who died in 1926. He later married Miss Lela Wilson, of Norfolk. There are three older sons, Edward, C. Lydon, Jr., and Gordon, and another son, Wilson, by the second marriage. Gordon has chosen a medical career, and is an oto-rhinolaryngologist in Norfolk.

From the beginning of his long and active medical career, he has been particularly interested in tuberculosis, and diseases of the chest have always been his sub-specialty in the field of internal medicine. He assisted Doctor Charles R. Grandy in pioneer tuberculosis free clinic work in Tidewater Virginia for 22 years, was Medical Director of Tidewater Victory Memorial Hospital (tri-county tuberculosis) during its lifetime, and has been in charge of the Tuberculosis Unit of the Norfolk General Hospital since its organization. He is also consultant in tuberculosis at the U. S. Naval Hospital, Portsmouth, Virginia. During World War I, he served with a Selective Service Draft Board of the City of Norfolk, and was Chairman of the Draft Boards in the City of Norfolk during World War II. He is a member of the Kiwanis Club, and has been on the Board of Stewards of the Ghent Methodist Church for a number of years.

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Many other organizations have profited by his active membership and his work for them. He has headed the Medical Staff of the Norfolk General Hospital, and has also given freely of his time and advice to the Staffs of the DePaul and Leigh Memorial Hospitals of Norfolk. He is a past-president of the Norfolk County Medical Society, and during his term in 1929, organized its permanent Annual Spring Clinic for Tidewater physicians. For the Medical Society of Virginia, he has served as a Vice-president, and as Councilor of the Second District, as well as heading the Committee on Tuberculosis for some years. He is a past president of the Alumni Association of the Medical College of Virginia. The Seaboard Medical Association elected him its President in 1943. He is a Diplomate of the American Board of Internal Medicine, a Fellow of the American College of Physicians, and a Charter Member of the American College of Chest Physicians, for which he is Governor of Virginia at this time.

This introduction of your new President would not be complete without stating that his physical stature is in inverse proportion to his capacity for useful work. His experience has been shared freely over the years with those asking it. Although alone in a large private and consulting practice, you will never find a committee meeting too small or too far for him, nor any medical or administrative problem too great for him to try to solve. We are fortunate in having him to guide our Society through the coming year. W. P. A.

Negroes and the VMS

THE refusal of the House of Delegates of the Medical Society of Virginia, which met here this week, to admit Negro physicians as members of the Society was softened somewhat by the subsequent adoption by unanimous vote of a resolution to invite Negro doctors to attend its "regularly scheduled scientific sessions", accompanied by a recommendation that its constituent groups adopt a like policy. Furthermore, the resolution directed the Society's professional publication, the Virginia Medical Monthly, to invite the submission of articles by members of the Old Dominion Medical Society (the State organization of Negro doctors) "for publication if and as merited".

As a matter of fact, the House of Delegates had voted 53 to 30 to permit Negroes to be admitted into the Medical Society, the vote in favor of the step falling only five votes short of the two-thirds necessary to amend the Society's constitution.

President-elect John T. T. Hundley, who will take office in October, 1951, in moving the adoption of the resolution to invite members of the Old Dominion Medical Society to attend scientific sessions of the VMS and to submit articles for publication in the Virginia Medical Monthly, expressed his belief that any immediate steps beyond those outlined in the resolution were "impractical" for the reason that the problem is "social and political" as well as "medical". That action on the resolution was unanimous is indicative of the fact that Dr. Hundley's view was shared by members of the Society generally.

While recognizing the social aspects of the problem and freely conceding that they are not to be lightly brushed aside, we cannot refrain from expressing the hope that the Society's refusal to let down the bars to Negro doctors will not be construed by the latter as intending the slightest reflection on their professional standing or excellent services as medical practitioners. It was not so intended, they can be sure.

Here in Roanoke, as elsewhere throughout the State, there are skilled and conscientious Negro physicians who are rendering an invaluable service to the public and to their profession and who are rightly held in the utmost esteem by their white colleagues, with whom they work in cordial understanding and mutual respect. The fact that a decisive majority of the Society's House of Delegates was in favor of granting them

admission into its membership is a significant indication of the trend of the times that can hardly be minimized.

The Negro is earning deserved recognition of his worth and attainments as a citizen and as a member of society and the barriers of unreasonable prejudice are rapidly coming down, though not all at once. And that to our way of thinking, is as it should be.

(Reprinted from *Roanoke Times*, October 11, 1950, by order of the Council of the Medical Society of Virginia)

Bibliotheca Obstetrica

RUSH, BENJAMIN

RUSH, BENJAMIN, (1745-1813): On the means of lessening the pain and danger of childbearing and of preventing its consequent diseases: in a letter to Dr. Edward Miller by Benjamin Rush, M. D. *Medical Repository* 6:26,1803.

——: An account of the late Dr. Hugh Martin's cancer powder, with brief observations on cancer. Trans. American Philosophical Soc.2:212,1786 (photostat).

——: An inquiry into the effects of ardent spirits upon the human body and mind, with an account of the means of preventing and of the remedies of curing them. 7th ed. *Boston, Manning and Loring.* 1812. 36 p.

——: Medical inquiries and observations. VIII, 206 p. *Philadelphia, Richard & Hall.* 1789.

——: Essays, literary moral and philosophical . . . *Phil. Thos. & Samuel F. Bradford.* 1798. 378 p. 20 cm. (M.L.)

——: Works . . . *2nd American ed. Phila. Thos. Dodson* 1794.5 v. 21 cm. (M.L.), Biography, The Autobiography of Benjamin Rush. Princeton University Press. 1948. Iconography, Radbill, Samuel X. Trans. and Studies, Coll. Physicians. Philadelphia. 16:73,1948. As an Obstetrician, Rucker, M. Price, Ann. Med. Hist.3rd series 3:487,1941.

That three Philadelphians should have written upon blood-letting as means of lessening pain in difficult labors in the early 19th Century is not so remarkable when we consider the background. It was the time when blood-letting was well nigh an universal remedy. Philip Syng Physick used this remedy to relax a patient with an old and irreducible dislocation of the shoulder and thus made its reduction easy and painless. Their eulogistic descriptions of the benefits of blood-letting are almost convincing, even today. It was only when the statistical methods of Louis were introduced in the next generation that bleeding disappeared from the armamentarium. Rush says that it was in 1795 that "he was led to suppose that blood-letting might be effectual in lessening the violence of the disease and pains of parturition." In his writings Rush always referred to pregnancy and labor as disease, but in his lectures to medical students he treated of them under physiology. Dewees had begun to make use of the method in 1789 and evidently influenced Rush who supplied the philosophy for the procedure. Peter Miller supplies the evidence, in his graduation thesis, that it was an accepted method of treatment.

Benjamin Rush is easily the best known man in American Medicine, and yet comparatively little is known of his obstetrical activities. He seems to have taken great interest in the subject throughout his professional life. His preceptor was Dr. John Redman who was, according to Dr. Bond, the best obstetrical practitioner in Philadelphia. On his return journey from Paris to London in 1768-9, Rush tells of his hearing a woman cry in the woods, and of his delivering her on a blanket under the trees and of the woman naming the baby for him. The numerous students' notes that are in existence in the various libraries all over the country show that he regularly lectured on obstetrics under the head of physiology.

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PRESIDENT'S MESSAGE

During these troubled and serious times it is with fear and trembling that I accept the honor and responsibility of being your President during 1950-51—fear that I might take one step in the wrong direction, making a severe mistake, or hurting someone's feelings; trembling for fear I might not have the strength, ability and courage to do the things that should and must be done. With your cooperation and God's help, we will carry on the very best we know how.

Under the leadership of Dr. W. C. Caudill, our president for the past year, a number of worthy objectives have been accomplished.

The membership has been increased to 2205. Two or more local medical societies have been formed. It is believed that more frequent meetings in some local medical societies have been arranged. The number of Women's Auxiliaries has been increased. A move has gotten under way to provide adequate space for our headquarters and to employ the necessary personnel.

The Society's support of, and opposition to, legislation, according to its merit has been effective. We could continue with further references to progress of the year, but I shall refer later to some of these in my address.

This being a legislative year, a number of items of interest to the medical profession might be mentioned: an increase in appropriations for public health, especially for tuberculosis treatment and control, and for mental hygiene. When these proposed plans are completed, the facilities for each will be greatly improved and nearly doubled in capacity.

Legislation concerning the office of Chief Medical Examiner and the State Coroner system was improved, and an attempt to transfer this to the State Police Department was defeated.

The problem of regulating the practice of the healing art without a license, with special reference to the chiropractor, was definitely improved. This legislation passed both houses without a dissenting vote. References to such matters as the practice of chiropodists, practice by clinical psychologists, a resolution opposing socialized medicine, etc., are among other matters that may be found in the report of the Legislative Committee.

In Congress, a number of bills have been introduced, which, if passed, would lead to the Federal control of the practice of medicine. Practically all these bills thus far were either held up in committees or killed in the House or Senate. The Social Security Bill, otherwise known as HR6000, was passed. However, the part that concerned us most was deleted. Members of the medical profession were not included in Old Age pension insurance. The clause pertaining to permanent and total disability was also eliminated; had this last clause been left in the bill, it would have been unacceptable to the medical profession, and a tremendous expense to the Government. Much of this success was largely due to the efforts of the A.M.A. Department of Public Relations and the diligent hard work of a number of our most interested physicians.

The American Medical Association's officials, the Public Relations Department, through Whitaker and Baxter, and public relations' departments of our State societies have done, and are still doing a great job in trying to educate the public of the undesirableness of socialized medicine, advising the public of the disadvantages and hardships they would have to endure in case the Federal Government should gain control of the medical profession and hospitals. Your officers and paid personnel cannot do the whole job; it behooves each and every one to do his and her part.

There are many who feel that as soon as the elections are over and a new Congress convenes, many new bills in sheep's clothing will be introduced, endeavoring to destroy our medical independence. We must get together, keep together and work harder than ever before or our cause will be lost to us forever.

In December of last year, the A.M.A. raised its dues to $25 per member to help meet the increase in operating cost and to defray the expense of the educational campaign which it has conducted the past two years. Heretofore, they have been able to operate with fees received from the journals and advertisements. The cost of everything has more than doubled, including salaries; consequently, as they were not able to operate without additional funds, they had no recourse but to raise the dues. Many of the States have made an excellent showing, as high as 90 per cent of the membership of some states have paid their dues. We regret that Virginia

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has fallen way behind. Our Secretary-Treasurer informs me that as of October 1, less than 40 per cent of our membership have paid their dues. This is embarrassing and should be regarded as a discredit to our State Society. Organized medicine has done much for the individual membership in every State Society and for the profession; we must not let it down in its time of peril or greatest need.

Another phase should be considered; this Society has three Delegates to the A.M.A. We are entitled to one Delegate for each thousand paid members and fraction thereof. Unless we have more than a thousand paid members by December 31, 1950, we will stand to lose two Delegates. The responsibility of collecting A.M.A. dues has been assigned to the secretaries of the local medical societies. I strongly urge that action be taken by our society in the interest of improving the practice in this respect.

The Business Office or headquarters of our Society have been too small and inadequate for many years. Your preceding President, Dr. W. C. Caudill, appointed a Committee a few months ago to search for a suitable home, a place to house all the business offices for some time to come. This Committee has been at work. I am continuing that Committee, hoping they will meet with success in the near future.

We hear much about the increased cost of medical care. This increased cost is not due to professional services alone, but to increased cost of hospital services and drugs; in the final analysis, it all can be traced to increase in labor costs. What is the answer? Prepaid medical care and hospitalization. Every local society should encourage the sale of some form of prepaid medical care and hospitalization, whenever possible. I suggest that each local medical society appoint a committee to promote these plans.

The time has come when the public should feel free to discuss medical fees with his or her doctor. One has just as much right to inquire what an operation, delivery or any professional service will cost before entering into a contract or engaging a physician for certain services, as he has on entering a store and inquiring the price of any article for sale, and should be encouraged to do so. When this is done in a helpful manner, it will go a long way towards relieving hard feelings and misunderstandings concerning what the public considers over-charging. An approach to this should be made on a local level. A fee that is considered reasonable in Roanoke, Norfolk or Richmond might be considered exorbitant in other sections of the State; the expense differential seems obvious. This should be considered seriously by public relations committees, both State and local. I hope they will see fit to discuss it freely in the news and editorial columns the coming year.

I have been advised by one of the members of the Board of Medical Examiners that they are not at all satisfied with the present Medical Practice Act. I am also informed that we have one of, if not the best, Coroners System in this country, which is mainly a product of this Society. Why cannot we have an Examiners System second to none?

The present Medical Practice Act was passed in 1944. This legalized chiropractic and naturopathy as branches of the healing art. It also put two chiropractors and one naturopath on the Board of Medical Examiners. They examine their own men, but have equal standing of any other M.D. on the Board in all scientific discussions. They are enjoying much prestige from their association with scientific medicine as bestowed upon them through the law and point with great pride to the certificates on their walls signed by the President and Secretary of our State Board of Medical Examiners as M.D.'s. Some feel that there should be two separate and distinct Boards set up by the Legislature. This matter will be referred to our Legislative Committee and our attorney for study and action. Would those of you who are interested in this subject please discuss this or present your views on it to some member of the Legislative Committee. As the Legislature does not convene until 1952, you have a little over a year to study and lay your plans.

One of my main objectives for the coming year is a special assignment for the Tuberculosis Committee. There are reasons to believe that the tuberculous patients in our mental and penal institutions are not being properly handled to prevent a universal spread of the disease among the inmates and personnel of the institutions. I shall request this Committee to make a survey or inspection of the State mental institutions, penitentiaries and jails of the larger cities and make its report to this Society with recommendations. This plan has been discussed with one of the heads of our tuberculosis sanatoria and with the Health Commissioner.

I feel it my duty to discuss the position of the

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Negro physicians licensed to practice in the State. According to the State roster, there were 146 as of July, 1949. They are anxious to join our local and State Societies, which will make them eligible to membership in the American Medical Association. I feel that they are justified in their ambitions and request. There appears to be nothing in the Constitution or By-Laws of the local medical societies to exclude them from membership. They must qualify and be recommended by the Membership Committee, a two-thirds vote of the members present at any business meeting is necessary for election.

However, there is a clause in the State Constitution which states that only white physicians may become members. This puts the Negro physicians in our State at a great disadvantage. They cannot enter the big clinics for post-graduate work because they are not members of the A.M.A. They cannot join any of the special organizations for the same reason. I recently had two applications from two colored physicians in this State to join the American College of Chest Physicians; they had met all qualifications in training, clinical experience and post-graduate work, but had not been permitted to join their local and State Society which was a prerequisite. A few months ago, the President appointed a Committee, headed by Dr. Vincent W. Archer, to study this problem, suggesting a way to circumvent this clause and submit a report.

Recently I mailed a questionnaire to the 146 colored physicians licensed to practice in the State asking the following questions:

1. What preliminary education did you have before beginning to study medicine?
2. Where did you get your M.D. degree?
3. Did you serve an internship before entering practice?

I received 96 replies: 49 of these had either an A.B. or B.S. degree before going into medicine; 46 had at least two years of college training. All got their M.D. degree out of the State. All but 8 served an internship before starting the practice of medicine. Gentlemen, I dare say that if a poll were taken of our membership, the preliminary training would not average near as high.

There is another side to this question. As a public health measure we need more Negro physicians and we need to keep them better trained after they start to practicing medicine. The Negro population of Virginia, as of January 1, 1949, estimated by the University of Virginia's Bureau of Population and Economic Research, was 710,362; this is almost five thousand Negro people to each Negro physician, 4,865 to be exact. A small percentage of the more successful and prominent Negroes are treated by white physicians, but the vast majority are treated by Negro physicians or in free clinics at public expense.

There are several institutions in this State that admit only Negro patients, yet, they are served by white interns. If these were served by Negro physicians, it certainly would help give the Negro physician a better idea about tuberculosis and venereal disease control which is so prevalent among this group.

A post-graduate course given for two weeks each year at the Medical College of Virginia for Negro physicians is rendering a great service. I am told by Dr. Hamilton Francis, who has been President of that group since the course was started many years ago, that they have had an average attendance of 30. He states that this course has been a blessing to the Negro physician.

The Legislature this year appropriated $10,000 for ten scholarships to Meharry, provided that on graduation the physicians agree to practice medicine in the rural section of the State for such a period of time as he received this financial assistance. It naturally follows that we are obligated to give him a better professional standing in other states. Being physicians as we are, they should be treated as such. I refer this matter to the Department of Clinical and Medical Education for special consideration.

I wish to commend, and we should encourage the teaching program which is being conducted by the staffs of our general hospitals for interns and residents. All members of the visiting staffs should be invited to attend these clinic sessions. It would be well for the smaller hospitals throughout the State to start such a program, even though they occasionally had to invite outside physicians to conduct the clinic.

Of each member of the Medical Society of Virginia, I wish to solicit full cooperation in helping to carry on this program. Suggestions such as you can give are essential to a successful year. Anytime I can be of any assistance to any component society, or in the affairs of our profession, I should want you to call on me.

C. L. HARRELL, M.D., Norfolk
President, Medical Society of Virginia

EDITOR'S NOTE: Address by President in assuming office at the meeting of the Medical Society of Virginia, October 11, 1950.

SOCIETY PROCEEDINGS

The Augusta County Medical Association

Held its regular quarterly meeting at Garden Center, Gypsy Hill Park, Staunton, September 6. Following cocktails and dinner at 6:00 p.m., there was a short business session, after which "The Red Cross Blood Program" was presented by Dr. John Whitney, head of the Eastern Area Blood Program.

New officers of the Association are: President, Dr. Richard P. Bell, Jr., Staunton; vice-presidents, Dr. James L. Davis, Waynesboro, Dr. J. Granville Crawford, Staunton, and Dr. Harry F. White, Fishersville; secretary, Dr. William G. Painter, Mt. Sidney; treasurer, Dr. Boyd H. Payne, Staunton; and censor, Dr. Chas. L. Savage, Waynesboro.

Roanoke Academy of Medicine.

At the meeting of the Academy on October 2, reports of officers and committees for the past year were presented, after which the new officers were installed, including Dr. E. G. Gill as president, and Dr. S. F. Driver as secretary. Both are of Roanoke. Dr. Gill then gave his presidential address.

The Southwestern Virginia Medical Society

Held its regular Fall meeting September 28, at Governor Tyler Hotel, Radford, the president, Dr. Harry Hayter of Abingdon, presiding. There were fifty-eight in attendance at the afternoon program and one hundred and thirty-five at the banquet. Thirteen new members were admitted to the Society.

At the afternoon session, the following papers were presented:

Chronic Diseases—Dr. Fred Heath, Blacksburg.

The Surgical Correction of the Deformed Obstructed Nose—Dr. F. D. White, Roanoke.

Subarachnoid Hemorrhage from Intercranial Aneurysms—Dr. William P. Tice, Roanoke.

Following a business session at which routine matters were discussed, the following were elected officers for 1950-51: President, Dr. James P. King, Radford; vice-president, Dr. Andrew F. Giesen, Radford, who has served the Society for several years as secretary-treasurer; and his successor, Dr. Richard C. Potter, Marion.

A movie, The Balance of Food and Energy, was then presented by the Upjohn Company. A social hour preceded the banquet after which Dr. Parson of the Department of Internal Medicine at the University of Virginia spoke on Obesity, and Dr. Robert C. Lynch of the Department of Surgery, Tulane University School of Medicine, New Orleans, gave a talk on The Present Approach to the Adenomata of the Thyroid.

Bristol, Virginia, was selected as the meeting place for May 1951.

Williamsburg-James City County Medical Society.

The September 15 meeting of this Society was called to order by the President, Dr. Eleanor Beamer-Maxwell, after a social dinner at the King and Kay Tea Shop in Williamsburg. A matter of special importance presented was a wire from the President of the State Society for information as to Medical Reserve Officers and other doctors in the community, should it be necessary to call them into military service. The secretary had answered this. Following the business meeting, Dr. Carlton J. Casey, a member who is on the staff of the Veterans Administration Hospital at Kecoughtan, projected and discussed a series of color transparencies of eye grounds. A number of questions were asked in regard to these.

Dr. Granville L. Jones of Williamsburg is secretary.

The Wise County Medical Society

Held its regular bi-monthly meeting at Norton Hotel in Norton, September 20th. There were twenty-one members present for "Dutch" country style steak dinner and the program.

The Society raised the annual membership dues from $3.00 to $5.00 per year with $10.00 initiation fee for new members after January 1, 1951. Dr. W. J. McGee of Keokee was elected to membership.

Dr. Alexander McCausland of Roanoke very ably addressed the Society on asthma, cause and treatment, with some remarks on ACTH, and Cortisone. Slides added greatly to the interest. Besides Dr. McCausland, the Society has been fortunate this year in having as guest speakers: Dr. Roy Hoover, Roanoke, and Drs. Herbert Acuff and W. M. Overholt of Knoxville, Tennessee.

Wise County Medical Society is one of the most active component units of State Medical Society, west of Roanoke, its attendance averaging eighty per cent of its membership.

Dr. W. B. Barton of Stonega is president and Dr. J. D. Culbertson of Coeburn secretary.

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NEWS

The Medical Society of Virginia

Had a most excellent meeting at the Hotel Roanoke in Roanoke, October 8 to 11, under the presidency of Dr. W. C. Caudill of Pearisburg. There was a registered attendance of over six hundred physicians, practically two hundred ladies, and over a hundred exhibitors, making a total of more than nine hundred. Much credit is due Dr. George S. Hurt and his able committee for their splendid work in making this meeting most successful.

The business sessions were interesting, the papers of a high character, and the exhibits, both scientific and technical, were all well worthwhile. Three awards were made to those presenting scientific exhibits: Drs. J. W. Devine, Sr., and Jr., of Lynchburg; Dr. William R. Sandusky of the University of Virginia and to Dr. Lee E. Sutton of the Medical College of Virginia.

At the closing session of the Society, Dr. C. Lydon Harrell of Norfolk was inducted into office as the next president of the Society. Those elected by the House of Delegates to serve with him are: President-elect, Dr. John T. T. Hundley of Lynchburg; vice-presidents, Dr. George S. Hurt of Roanoke, Dr. S. K. Ames of Cape Charles, and William R. Pretlow of Warrenton; and secretary-treasurer, Henry S. Johnson (re-elected), Richmond. The only change in councilors was the election of Dr. Frank A. Farmer of Roanoke for the Sixth District, succeeding Dr. Hundley. Dr. J. Morrison Hutcheson of Richmond was re-elected as one of the delegates to the A.M.A., with Dr. Carrington Williams, Sr., of Richmond as alternate.

Dr. J. D. Hagood of Clover was selected as the general practitioner of the year to be nominated for that honor to the American Medical Association at its clinical session.

Virginia Beach was selected as the 1951 place of meeting.

The Southern Medical Association

Is to hold its annual meeting in St. Louis, November 13-16, under the presidency of Dr. Hamilton W. McKay of Charlotte, N. C. General headquarters will be the Kiel Municipal Auditorium, where all meetings, scientific and technical exhibits will be housed. Several affiliated associations will hold meetings conjointly. St. Louis has available for this meeting a large number of first class rooms. For help in getting located, however, you may write the Housing Bureau, Southern Medical Association, 911 Locust Street, Room 406, St. Louis 1, Missouri.

The subscription dinner will be held on Tuesday. This will be followed by entertainment and a dance and the evening will be given over to pleasure. All activities at the Kiel Auditorium will be available to physicians who are members in good standing of their local and state medical societies, upon presentation of their membership cards. There is no registration fee.

The A.M.A. Clinical Session

Which will be held in Cleveland, December 5-8, is designed primarily for the general practitioner. The scientific sessions and the scientific and technical exhibits will be presented in the Cleveland Municipal Auditorium. Meetings of the House of Delegates on December 5 and 6 will be at the Statler Hotel and are open to all members and visitors in related fields.

Clinical sessions will be limited in attendance so that general practitioners may enter into the discussions and inquire about their own problems. The scientific exhibits will offer special demonstrations and the technical exhibits will feature the latest developments in drugs, equipment, books and allied medical products. Television will have a prominent place on the program.

The annual General Practitioner Award will be presented to the doctor selected by the House of Delegates from nominations made by State medical societies.

Last year's Clinical Session in Washington attracted over 4,000 doctors from every part of the United States and an increased attendance is expected this year.

Preceding the Clinical Session of the American Medical Association, a Public Relations Conference will be held at Hotel Statler, Cleveland, on December 3 and 4. The program calls for four work sessions—two each day. In attendance at these sessions should be some 300 doctors who are chairmen of State and county medical society Public Relations

committees, executive secretaries and Public Relations directors, officers of the A.M.A. Woman's Auxiliary, and representatives of other allied health organizations.

The Southern Electroencephalographic Society

Will meet in Williamsburg on November 27th and 28th. This meeting is in association with the Southern Psychiatric Society and will be held at the Williamsburg Inn. The presiding officer of the Society is Dr. Samuel C. Little. All local doctors interested in EEG are invited to attend the scientific session.

Medical College of Virginia News.

The School of Medicine in September matriculated 363 students, 84 in the first year class. All but 10 are Virginians. The upper class enrollment is somewhat larger than usual. Total college enrollment in all schools and departments, graduate and undergraduate, is 1419.

Dr. and Mrs. Everett I. Evans, Dr. and Mrs. George Z. Williams, Dr. Ernst Fisher and Dr. Robert Waller attended important scientific meetings and conferences in Europe during the past summer.

Dr. Isaac A. Bigger, professor of surgery at MCV, was paid tribute by 40 of his former residents for his 20 years of service to the college in an impressive and delightful program on September 22. The ceremony was held in Baruch Auditorium.

At this time a portrait of Doctor Bigger was presented to the college. The painting was done by Charles Hopkinson of Massachusetts. Dr. W. T. Sanger, president of MCV, accepted the portrait which will likely be hung in George Ben Johnston Auditorium.

A refrigerator for his office and a radio, television, phonograph combination were presented to Doctor Bigger by his former residents. Representatives of the Department of Medicine presented him with a set of silver goblets.

World Health Organization during the past summer referred two visitors in this country to the college for study and observation, Dr. Chavali Vyag of India in the Physical Medicine Department and Dr. Ernesto Saldias of Chile in the Department of Pediatrics, section on poliomyelitis. Dr. Randolph Batson of Vanderbilt University Medical School also looked in on the work in poliomyelitis recently.

Construction has started on a new addition to the Brown-Sequard Laboratory which is used for experimental work associated with animal studies.

The Richmond Eye Hospital, including ear, nose and throat services, will shortly go under construction in the 400 block North Twelfth Street. Tunnel connection with the rest of the college has almost been completed. This hospital will be under a separate self-perpetuating board but has a long-term contract of affiliation with the college.

The college library circulated 4,522 more books last year than the year before. Such books are loaned widely to members of the profession over the State as well as being circulated in Richmond.

Benjamin S. Andrews, director of speech and hearing rehabilitation for the Virginia Society for Crippled Children, now has offices located in Memorial Hall, formerly Memorial Hospital. His service will be State-wide as well as local.

The handsomely remodeled residence at 1006-08 East Marshall Street, lately occupied by senior nurses of the college, has been named Nannie Minor House for a beloved Virginia nurse, alumnus of the college, and daughter of the distinguished law professor, John B. Minor, of the University of Virginia.

President W. T. Sanger reported gifts, grants and bequests for the year ending June 30, 1950 of $847,445.46; these items for the first two and one half months of the current fiscal year amounted to $99,526.63, all exclusive of State tax funds.

The Graduate Lecture Program

Of the University of Virginia Medical School was commenced on Monday evening, September 25. The program is for House Officers and the Staff of the University of Virginia Hospital, and is open to Virginia physicians without charge. There will be two series of twelve lectures each, the first being from September 25 through December 11. There will then be an intermission of seven weeks on account of the Christmas recess and mid-year examinations, the

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second series starting on February 5. Unless otherwise indicated, all lectures will be given at 8:00 p.m., in the Nursing School auditorium, McKim Hall. Further information desired may be obtained from Dr. Douglas E. Bragdon, corresponding secretary for the Lectures.

Lectures yet to be held in the first series with the guest speakers are:

November 6—Dr. W. C. Hueper, Cancer Control Branch, U. S. P. H. S.—Industrial Carcinogenesis

November 13—Dr. Shields Warren, Director, Division of Biology and Medicine, U.S. Atomic Energy Commission—The Acute Radiation Syndrome and Its Therapy

November 20—(Alpha Omega Alpha Lecture) Dr. Chester Jones, Massachusetts General Hospital—The Problem of Nutrition After Gastroenterological Surgery (tentative subject)

November 27—(Staige D. Blackford Memorial Lecture) Dr. Robert S. Palmer, Massachusetts General Hospital—The Rationale of Current Therapy for Essential Hypertension

December 4—Dr. Joseph E. Moore, The Johns Hopkins School of Medicine—An Evaluation of Public Health Measures for the Control of Syphilis

December 11—Dr. John H. Gibbon, Jr., Director of Surgical Research, Jefferson Medical College, Philadelphia—The Present Status of Mechanical Heart and Lung Machines

Dr. Hiram W. Davis,

Class of '42, Medical College of Virginia, has been appointed superintendent of Huntington (W. Va.) State Hospital, to fill the vacancy caused by the death of Dr. E. F. Reaser last July. Dr. Davis has recently been at McGuire Veterans Administration Hospital, Richmond.

Dr. William E. Apperson,

For some time a member of the staff of Blue Ridge Sanatorium, Charlottesville, has accepted the position as medical assistant to the State director of the division of tuberculosis control, and is now making his home in Richmond.

New Medical Chief at Kecoughatan.

Dr. Paul Baird has been named chief of professional services at Kecoughatan Veterans Home near Hampton, succeeding Dr. Merrill Fox, who has been transferred to Lake City, Florida. Dr. Baird is a 1935 graduate of Tulane Medical School and has been transferred from Baltimore.

Fellowships in Industrial Medicine.

The Institute of Industrial Health of the University of Cincinnati will accept applications for a limited number of fellowships which are being offered to qualified candidates who wish to pursue a graduate course of instruction which will qualify them for the practice of Industrial Medicine. Candidates who complete satisfactorily the course of study will be awarded the degree Doctor of Industrial Medicine.

Any registered physician, who is a graduate of a Class A medical school and who has completed satisfactorily two years of residency (including internship) in a hospital accredited by the American Medical Association may apply for a Fellowship in the Institute of Industrial Health. The course of instruction consists of a two-year period of intense preliminary training in the basic phases of Industrial Medicine followed by one year of practical experience under adequate supervision in industry. During the first two years, the stipends for the Fellowships vary from $2,100 to $3,000. In the third year the candidate will be compensated for his service by the industry in which he is completing his training.

Requests for additional information should be addressed to the Institute of Industrial Medicine, College of Medicine, Cincinnati 19, Ohio.

Tuberculosis Association News.

Dr. Charles P. Cake of Arlington was elected president of the Southern Trudeau Society and Miss Nora Spencer Hamner of Richmond was elected president of the Southern Tuberculosis Conference at the recent meeting held in Roanoke.

Representatives from 15 Southern States and the District of Columbia attended the three day meeting for discussion of the public health, medical and social aspects of tuberculosis and one session on the financing of voluntary tuberculosis associations through the sale of Christmas Seals.

Miss Hamner, the first Virginia woman to hold the office of president of the conference, announced the official opening of the 44th annual Christmas Seal Sale as of November 20.

Dr. John F. Woodward,

Who practiced ophthalmology for many years in

Norfolk until his retirement in 1942 to locate in Ivanhoe, North Carolina, has returned to Virginia and is now living at Virginia Beach.

Dr. Frank B. Truesdell

Has been elected president of the Richmond Choral Society, an organization in which he has always taken an active part.

Dr. Richard A. Bagby,

Class of '43, Medical College of Virginia, announces that he is located at 509 Bay Street, Tampa, Florida, for the practice of otolaryngology.

The Society for Investigative Dermatology,

At its last annual meeting, elected the following officers for the coming year: President, Dr. Marion B. Sulzberger, New York; Vice-President, Dr. Nelson Paul Anderson, Los Angeles; Secretary-Treasurer, Dr. Herman Beerman, Philadelphia; Director, Dr. Chester N. Frazier, Boston.

At this meeting, the Society selected Atlantic City as the place for their next annual two-day meeting in 1951.

Southeastern Allergy Association.

The sixth annual meeting of this Association will be held at the Vinoy Park Hotel, St. Petersburg, Florida, January 20-21, 1951. Reservations are to be made individually with the Hotel, which is on the American plan.

A panel discussion of Allergic Chest Diseases will be under the direction of Dr. Raymond Arp. Dr. Clarence Thomas will head the panel on Rhinological Allergy. Dr. Harold Abramson, president-elect of the American College of Allergists, and Dr. Horace Baldwin, vice-president of the American Academy of Allergy, have been invited to be guest speakers.

There will be a "Stump the Experts" program this year. Dr. Clarence Laws will be the "ring master". Bring your "problems" or "$64.00 questions" to the meeting and hear what the "experts" have to say.

A cocktail party and banquet will be held at the Vinoy Park Hotel. Further information may be had from the secretary, Dr. Katharine Baylis MacInnis, 1515 Bull Street, Columbia, S. C.

News from the University of Virginia Department of Medicine.

Dr. Albert A. Fisk has joined the faculty of the Department of Medicine, University of Virginia, as assistant professor of biochemistry. Dr. Fisk is a graduate of Harvard College and the School of Medicine, Yale University, where he held the Junior Brown Fellowship in Pathology. In 1948-1949, he was American Cancer Society Fellow at the Massachusetts Institute of Technology and, during the past year, he has been engaged on studies in protein chemistry under Dr. Linus Pauling at the California Institute of Technology.

Dr. Grover C. Pitts has been appointed assistant professor of physiology at the Department of Medicine, University of Virginia. Dr. Pitts received his Ph.D. at Harvard University in 1943 and has been associated with the Harvard Fatigue Laboratory and the Naval Medical Research Institute.

Construction has been started on an addition to the Department of Medicine, University of Virginia, which will provide additional laboratory space for the Departments of Biochemistry, Pharmacology, Physiology, Pathology, Microbiology, Internal Medicine and Surgery, as well as new animal quarters, classrooms and an auditorium to seat 240. The total project, including equipment, will cost approximately $950,000. Construction of a unit for cancer teaching and research, costing approximately $250,000 is also under way.

Dr. Dudley C. Smith, Professor of Dermatology and Syphilology, University of Virginia, died on August 30th.

Dr. Cary N. Moon, Instructor in Otolaryngology at the University, has been granted a leave of absence for military service.

The following grants from the National Institute of Health have been recently received by the Department of Medicine, University of Virginia; $24,829 for cancer teaching; $4,968 for studies on the effects of hyaluronidase upon the course of tuberculosis in rabbits treated with streptomycin, under Drs. J. R. Cash and S. W. Britton; $2,592 for studies on trace elements in human blood, under Dr. B. S. Leavell; $9,093 for studies on the effect of CNS depressants on metabolism, under Dr. C. L. Gemmill; $17,500 for the training program in psychiatry, under Dr. D. C. Wilson.

The Piedmont Proctologic Society,

Composed of specialists from Virginia, North and

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South Carolina, Tennessee and Georgia, at its annual meeting in Hendersonville, N. C., August 26, elected Dr. C. R. Deeds of Hendersonville as its new president; Dr. J. M. Stockman of Knoxville, Tenn., was made vice-president; and Dr. C. S. Drummond of Winston-Salem was re-elected secretary-treasurer. The next meeting of the Society will be on March 31, 1951 at Knoxville, Tennessee.

Prize Essay Contest.

The American Dermatological Association is offering a prize of three hundred dollars for the best essay submitted of original work, not previously published, relative to some fundamental aspect of dermatology or syphilology.

Manuscripts typed in English with double spacing as for publication, together with illustrations, charts and tables, are to be submitted in triplicate not later than February 1, 1951. Further information regarding the essay may be obtained from Dr. Louis A. Brunsting, Secretary, American Dermatological Association, 102-110 Second Avenue, Southwest, Rochester, Minnesota.

On City Board of Health.

Dr. M. Pierce Rucker has been elected chairman of the Richmond Board of Health, succeeding Dr. B. H. Martin, resigned. Dr. Harry J. Warthen has been named to fill Dr. Martin's unexpired term.

Dr. James A. Smith,

A native of Ohio and a graduate of the George Washington Medical School, has been appointed in charge of the Page-Warren-Shenandoah Health District and has entered upon his duties with offices at Luray. He was recently with the U. S. Public Health Service in Galveston, Texas.

Basic Science Board.

Governor Battle has appointed Dr. Carl Speidel of the University of Virginia, and Dr. Frank Apperly and Dr. Sidney Negus of the Medical College of Virginia, as members of the Basic Science Board of the State Board of Medical Examiners. They will furnish examinations for the licensing of chiropractors and naturopaths in Virginia.

The American College of Chest Physicians

Offers a cash prize award of $250.00 to be given annually for the best original contribution, preferably by a young investigator, on any phase relating to chest disease. The prize is open to contestants of other countries as well as those residing in the United States. The award will be made at the next annual meeting of the College in Atlantic City, June 7-10, 1951.

Detailed information about the contest may be obtained from the Executive Office of the College, 500 North Dearborn Street, Chicago 10, Illinois.

Office Available.

Doctor's or dentist's office available in Richmond immediately. Corner Harrison and Franklin Streets. Call 5-4397. (*Adv.*)

Convalescent Home.

"Durell Haven", Arrington, Nelson County, Va., open for a limited number of convalescent or elderly people. All modern conveniences. Write Laura B. Horsley, R. N., Arrington, Va. (*Adv.*)

Wanted—

Relief doctor for a full month. Must have Virginia license. House, jeep and board furnished with attractive salary. Must be sober. Write William A. Cover, M.D., Big Rock, Va. (*Adv.*)

Wanted Contract Practice.

Physician desires contract practice in either industrial section or coal mining area with guaranteed salary and opportunity for developing private practice. Give particulars in first letter. Address "Contract Practice", care *Virginia Medical Monthly*, 1200 East Clay Street, Richmond 19, Va. (*Adv.*)

OBITUARIES

Dr. Henry Martin Snead,

Columbia, died October 8, after an illness of several months. He was sixty-five years of age and a graduate in medicine from the former University College of Medicine, Richmond, in 1907. Dr. Snead had practiced in Petersburg for a number of years before giving up private work and accepting the position as health officer with the State Department of Health. He was a former president of the Petersburg Medical Faculty, a member of the Fourth District Medical Society and was for some years a member of the Medical Society of Virginia. His wife, two daughters and a son survive him.